This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

COUNTWAY LIBRARY
HC 1H1I N

S.R.B.

15. E. 205

E. F. Mahady Co.,
Medical Books,
Boston.

THE EARLY DIAGNOSIS OF HEART FAILURE

PRINTED BY
SPOTTISWOODE AND CO. LTD., LONDON
COLCHESTER AND ETON

THE EARLY DIAGNOSIS OF
HEART FAILURE

AND OTHER ESSAYS
ON THE
HEART AND CIRCULATION

BY
T. STACEY WILSON, M.D. (EDIN.)
F.R.C.P. (LOND.), B.SC. (EDIN.)
SENIOR PHYSICIAN, GENERAL HOSPITAL, BIRMINGHAM
LECTURER ON MEDICINE IN THE DENTAL SCHOOL OF THE UNIVERSITY
OF BIRMINGHAM

WITH ILLUSTRATIONS

NEW YORK
WILLIAM WOOD AND COMPANY
MDCCCCXV

BOSTON MEDICAL
NOV 5 1916
LIBRARY

2 N 57 A 32

4.05

PREFACE

This volume consists of separate essays dealing mainly with Heart Failure.

Its aim throughout is to help the practitioner, when at the bedside of his patient, by suggesting to him new methods of observation which give fresh insight into the mode of the heart's action in health and disease.

The book traverses ground that is more or less new, since it recognises types of cardiac failure which are often ignored (in their early stages at least), and indicates certain methods of clinical diagnosis—some of them simple—which do not receive full description, and in several instances are not treated of at all, in the ordinary text-books upon the heart.

For example, such conditions as 'high diaphragm' and 'diminution in the area of liver dullness' are rarely looked for as signs of diminution of the total volume of blood in circulation; nor is this lessened volume of circulating blood generally recognised as a sign of myocardial weakness.

Therefore, in advancing new theories, advocating new methods, and dealing with matters not hitherto regarded as of proved clinical importance, it is incumbent on the author to bring forward at the same time adequate clinical evidence in their favour. Hence the numerous diagrams and details of cases which are introduced.

Practically no reference is made to the work of other observers, and for this defect the writer would fain apologise. But, considering that he has found it difficult to compress within reasonable limits the results of thirty years' clinical observations upon the heart, it is evident that the introduction and discussion of the work of others would have unduly increased the size of the present volume.

As the essays have been written at various times during the last twenty years, and deal with subjects closely related to each other, it is inevitable that there should be a certain amount of repetition; no attempt has been made to remedy this, the writer preferring that each essay should remain individually complete.

At the end of the book will be found a series of summaries of the points raised in connection with each of the main subjects, and references are given to the pages dealing with the various subjects.

For instance, the subject of the distensibility of the heart wall is discussed in connection with its value for classification, in connection with heart failure in adolescence in adult life and in later life, and also in connection with its relationships to heart failure with and without dilatation.

These various points are brought together when the subject of 'Distensibility' is summarised at the end of the book, and references are given to the pages in the body of the work, where the subject is discussed more fully. The same is true of 'Heart Failure with Enlargement,' 'Heart Failure without Enlargement,' 'Compensatory Diminution of the Blood Volume,' &c.

Some of the less important subjects are summarised in the subject index at the end of the volume.

It is the author's hope that this book will not only prove of direct clinical value, but also that it will stimulate others to explore and develop new fields of clinical enterprise in connection with the heart and circulation.

In conclusion, the author desires to acknowledge his indebtedness to Dr. Mary Sturge for many helpful suggestions; to his resident physicians, one and all, for valuable aid in collecting clinical material in his wards at the Birmingham General Hospital; and to Dr. Leonard G. J. Mackey for much kind assistance and many suggestions in revising proofs.

BIRMINGHAM:
November 4th, 1914.

CONTENTS

PART I.—ESSAYS DEALING MAINLY WITH THE EARLY DIAGNOSIS OF HEART FAILURE

Essay 1.—EARLY DIAGNOSIS OF HEART FAILURE.

PAGE

Introductory—Classification—Distensibility of the heart in adolescence 1

EARLY DIAGNOSIS OF HEART FAILURE WITH ENLARGEMENT:

1. In DISTENSIBLE hearts
    Symptoms—Physical signs—Theory and method of percussion—Auscultation of the heart—How to detect the first signs of cardiac failure 12

2. In hearts of MODERATE DISTENSIBILITY
    Distensibility of the heart in adult life—Symptoms and physical signs 31

Changes in the left ventricle in heart failure with enlargement . 46

EARLY DIAGNOSIS OF HEART FAILURE WITHOUT ENLARGEMENT:

Theoretical considerations:
    In DISTENSIBLE HEARTS, i.e. HEART FAILURE WITH SMALL HEART 50

Symptoms and physical signs 54
    Diminution in the size of the heart a sign of muscular failure in valvular disease 62

EARLY DIAGNOSIS OF HEART FAILURE IN THE RELATIVELY RIGID HEART OF LATER LIFE.

Introductory—Modifications in the signs and symptoms of heart failure which result from the increasing rigidity of the tissues in later life 70

Distensibility of the heart in later life 76

PAGE

Heart Failure in relatively Rigid Hearts:
- 1. With enlargement 85
- 2. Without enlargement in moderately strong hearts . . 86
- 3. Without enlargement in decidedly weak hearts . . 91

Illustrative cases—Heart failure without enlargement complicating pneumonia—With and without enlargement in two patients over eighty years of age 97

In cases of valvular disease in later life 110

Diagnostic difficulties associated with the diagnosis of heart failure in later life as regards: Dyspnœa—Palpitation—Faintness—Anginal pain 112

Diagnostic value of venous phenomena in later life . . . 132

Recognition of arterial resistance as a factor in heart failure—Recognition of the degree of rigidity of the heart wall—Evidences of rigidity in valvular disease—Difficulties when one ventricle is weak and the other is strong 142

Essay II.—ON THE VALUE OF AN ABNORMAL RISE IN THE LEVEL OF THE DIAPHRAGM AS A GUIDE TO THE AMOUNT OF BLOOD IN ACTIVE CIRCULATION.

High diaphragm due to malnutrition in enteric fever—Due to starvation in the treatment of gastric ulcer—After severe hæmorrhage—In anæmia—In neurasthenia—High diaphragm in heart weakness 163

Essay III.—DIMINUTION IN THE AREA OF THE LIVER DULLNESS AS AN IMPORTANT SIGN OF MYOCARDIAL WEAKNESS OR OF DEFICIENCY IN THE TOTAL VOLUME OF THE BLOOD.

Compensatory diminution in the blood volume in heart weakness and its effect on the liver—Variations in the size of the liver dullness as detected by percussion 185

Diminution of the liver dullness due to myocardial weakness—Due to a shrinkage in the total volume of the blood from hæmorrhage, from anæmia, from deficiency of essential constituents of the blood 195

Essay IV.—ON SOME DIAGNOSTIC DIFFICULTIES ASSOCIATED WITH DILATATION OF THE RIGHT VENTRICLE

Various types of difficulty—Aneurysm of the transverse arch of the aorta simulating upward dilatation of the right ventricle . 209

PAGE

Difficulties associated with the recognition of the pulmonary and aortic systolic murmurs—Of the murmurs due to tricuspid regurgitation, the true tricuspid systolic, the superior vena cava murmur, and the murmur sometimes produced at the venous valves—Of the arterial compression murmur . . . 215

PART II.—ESSAYS DEALING MAINLY WITH DILATATION OF THE HEART DUE TO OVERSTRAIN

ESSAY V.—CONDITION OF THE HEART IN THE ANÆMIA OF ADOLESCENCE.

Dilatation of the heart upwards and to the left—Statistics—Dilatation due to upward increase of the right ventricle—Displacement of the apex upwards—Dilatation to the right—Failure of the left ventricle—Cause of upward dilatation—Pathological aspects of upward dilatation 227

THE PULMONARY SYSTOLIC MURMUR—Its mode of production . 254

Other auscultatory phenomena—Right ventricular third sound and mid-diastolic murmur 279

Cases illustrating cardiac dilatation in the anæmia of adolescence . 287

ESSAY VI.—THE HEART IN OVERSTRAIN.

Cases of overstrain in adolescence—In adult life—In later life—Case of overstrain resulting in lasting tachycardia 308

ESSAY VII.—TWO CLINICAL LECTURES ON DILATATION OF THE RIGHT VENTRICLE, DELIVERED 1894.

This early essay is practically a summary of Essay V. and of parts of Essay I. 329

ESSAY VIII.—ON DISPLACEMENT OF THE HEART.

By CHANGES WITHIN THE HEART ITSELF—Aneurysm—Aortic regurgitation a cause of downward displacement 345

UPWARD DISPLACEMENT OF THE APEX in anæmic dilatation of the right ventricle—Statistics of its frequency—Its relationship to the age of the patient—To the amount of upward dilatation—To the presence of dilatation of the ventricle to the right—To failure of the left ventricle—Cause of upward displacement of the apex 348

DISPLACEMENT BY EXTRINSIC AGENCIES 361

- Downwards by abnormal lowness of the diaphragm in emphysema, &c.—In chronic venous congestion of the lungs . . . 362
- Displacement upwards by rise of diaphragm 368
- Lateral displacement of the heart 371
- Altered relationships of the heart when displaced . . . 379

PART III.—ESSAYS DEALING WITH VALVULAR DISEASE, ESPECIALLY IN RELATION TO VENTRICULAR EXPANSION

ESSAY IX. PAGE

THE NATURE OF THE EXPANSION PHASE OF THE CARDIAC CYCLE AND THE PHENOMENA ASSOCIATED WITH IT 384

This essay is mainly a summary of the three following essays :

ESSAY X.

The DIASTOLIC EXPANSION MOVEMENT of the ventricles as A FACTOR IN COMPENSATION for disease of the mitral valve

Cardiographic and clinical evidence as to the movements of the heart 400

ESSAY XI.

On the THEORY OF COMPENSATION in incompetence and stenosis of the mitral valve 412

ESSAY XII.

On the diastolic cardiac sound which causes spurious reduplication of the second sound at the apex and is sometimes called THE THIRD SOUND OF THE HEART 427

ESSAY XIII.

On THE CONDUCTION of the mitral systolic murmur DOWN THE SPINE : its diagnostic and prognostic value 442

PART IV.—SUNDRY ESSAYS BEARING UPON THE DIAGNOSIS OF HEART FAILURE

ESSAY XIV.—THE DIAGNOSTIC AND PROGNOSTIC IMPORTANCE OF PALLOR AS A SYMPTOM OF HEART DISEASE AND HEART FAILURE.

Distinction between anæmia and pallor—Pallor in transient and persistent heart weakness—Cases showing pallor as a sign of faulty compensation 448

ESSAY XV.—SOME LEG PAINS OF CIRCULATORY ORIGIN

LEG PAINS DUE TO DILATED DEEP VEINS OF THE THIGH :
Pathology—Symptoms—Referred pain—Neurasthenia—Illustrative cases—Treatment 459

PAGE

Leg Pains due to Deficient Blood Supply:
  Due to feebleness of the general circulation—A sign of myocardial weakness 467
  Due to local causes of interference with the blood supply—Cramp — Intermittent claudication — Due to vasomotor disturbance 471

Essay XVI.

On the value of reverberation in the diagnosis of the size and position of the stomach 480

Essay XVII.—A CASE OF ARHYTHMIA WHICH THROWS NEW LIGHT UPON THE VENOUS PULSE AND THE ACTION OF THE AURICLES.

Summary of the argument—Interpretation of cardiograms—Mechanical stimulation of the ventricle a cause of extra systoles—Tonic activity of the auricles and great veins—Work done by the extra systole—Sudden distension of the auricle a cause of an auricular contraction—True contraction of the veins—Interpretation of tracings 489
Summary of Facts and Theories here Brought Forward . 523
Physiology of the Auricles and Veins in the Light of this Research 528

SUMMARIES

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH THE DISTENSIBILITY OF THE HEART WALL

Factors which determine the degree of distensibility of the heart wall—Variation in the distensibility of the heart according to the age of the patient 533

Distensibility of the heart in relation to heart failure without enlargement 537

Clinical estimation of the distensibility of the heart 539

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH HEART FAILURE WITH ENLARGEMENT

I. Theoretical Considerations:
  Factors which determine the amount and type of cardiac dilatation 542

PAGE

Various types of dilatation of the heart:
1. Adult type 543
2. Adolescent type 543
3. Intermediate types 545
Overstrain and its relationship to cardiac dilatation—Apportionment of strain between the two ventricles—Causes of overstrain 545

II. PATHOLOGICAL ASPECTS OF DILATATION OF THE RIGHT VENTRICLE IN ADOLESCENCE AND EARLY ADULT LIFE 546

III. CLINICAL ASPECTS OF HEART FAILURE WITH ENLARGEMENT.

1. In distensible hearts, i.e. the ADOLESCENT OR UPWARD TYPE of dilatation of the right ventricle
Clinical features in general—Changes in pulmonary second sound—Pulmonary artery systolic murmur—Third sound, &c. 547
2. In the moderately distensible heart of adult life 557
Physical signs in connection with the right ventricle, i.e. THE ADULT TYPE of dilatation 558
Clinical features in general:
Phenomena due to tricuspid regurgitation . . . 559
Venous engorgement—Tricuspid systolic murmur—Superior vena cava murmur—Murmur arising at the venous valves 559

SUMMARY OF PHYSICAL SIGNS IN CONNECTION WITH THE LEFT VENTRICLE 561

HEART FAILURE WITH ENLARGEMENT IN RELATIONSHIP TO VALVULAR DISEASE 562

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH HEART FAILURE WITHOUT ENLARGEMENT

General considerations in connection with heart failure without enlargement—In the distensible heart of adolescence—namely, HEART FAILURE WITH SMALL HEART—In the less distensible heart of adult and later life 564

Clinical features of heart failure with small heart—In less distensible hearts 567

Enlargement of a small heart a sign of returning strength . . . 570

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH EARLY DIAGNOSIS OF HEART FAILURE IN LATER LIFE

EARLY DIAGNOSIS MORE DIFFICULT BECAUSE : PAGE
I. Changes in the size of the heart indicative of failure take place less readily owing to the increasing 'rigidity' of its tissues 571
II. Changes indicative of pathological variations in the volume of blood in the thoracic viscera and in the liver take place less readily owing to the increasing rigidity of the fibrous tissues of the body 571
III. There are difficulties in appraising the true value of changes in the strength of the blood pressure owing to the frequency of a pathological increase in the arterial resistance . . 572
IV. There are difficulties associated with the occurrence of myocardial weakness due to disease of the coronary arteries 573
V. There are difficulties due to the fact that greater dependence has to be placed upon general symptoms of heart failure, and that they are often misleading: such are breathlessness, palpitation, faintness, anginal pain . . . 575
Summary of evidences of heart failure in later life—Summary of venous phenomena—of opposing and apparently contradictory statements 576

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH COMPENSATORY DIMINUTION OF THE BLOOD VOLUME.

COMPENSATORY DIMINUTION OF THE TOTAL VOLUME OF THE BLOOD OWING TO A DEFICIENCY OF ONE OR MORE OF ITS ESSENTIAL CONSTITUENTS 577

In starvation—In lack of water (cholera, &c.)—In hæmorrhage—In neurasthenia, &c. 578

COMPENSATORY DIMINUTION OF THE VOLUME OF BLOOD IN ACTIVE CIRCULATION OWING TO CARDIAC INEFFICIENCY 578

Clinical signs given by diminution of blood in the thoracic viscera, i.e.:
1. Rise of diaphragm (evidenced by rise in the gastric resonance and the liver dullness and retraction of the supra-clavicular hollows) 579
2. Diminution in the size of the heart 581
3. Diminution in the amount of distension of the liver (shown by decrease in its area of absolute dullness) . . . 581

PAGE
4. Emptiness of the auriculo-venous reservoir (shown by changes in the pulsation palpable in the jugular bulb . 583
5. Emptiness of the veins 583

SUMMARY AND INDEX OF FACTS AND ARGUMENTS IN CONNECTION WITH THE AURICULO-VENOUS RESERVOIR.

ANATOMICAL CONSIDERATIONS :
Constitution of reservoir 584
PHYSIOLOGICAL CONSIDERATIONS :
Tonic activity of its walls (both auricle and veins) and maintenance of definite blood pressure within it—Contractile activity of the walls of the auricle and the veins—Tonic activity of the walls of the right ventricle during its relaxation phase 584
Physiological points in connection with the venous valves, the superior vena cava, the azygos vein 587
PATHOLOGICAL CHANGES IN THE RESERVOIR AND CLINICAL PHENOMENA ASSOCIATED WITH THEM :
Dilatation of auricle, of veins, &c. 588
CLINICAL PHENOMENA SHOWING THE CONDITION OF THE RESERVOIR :
Pulsation in the jugular bulb—Signs of over-fullness or emptiness of the blood-vessels of the thoracic viscera and of the liver 589

SUMMARY AND INDEX OF ARGUMENTS AND FACTS RELATING TO COMPENSATORY PHENOMENA IN CONNECTION WITH THE HEART AND BLOOD-VESSELS.

Phenomena connected with the veins and arteries—With the heart—Compensation in mitral regurgitation—In mitral stenosis . 592

SUBJECT INDEX 597

ILLUSTRATIONS

FIGS. PAGES

1–5. A Case in which the Occurrence of Gradual Cardiac Enlargement Indicated Returning Strength after Myocardial Weakness 66, 67

6. A Case where the Adolescent Type of Dilatation occurred in Late Adult Life 84

7–15. A Case of Severe Pneumonia with Myocardial Weakness, and where Variations in the Cardiac Strength were shown by Daily Variations in the Size of the Cardiac and Liver Dullness and in the Level of the Diaphragm 100, 101, 103

16–18. Small Heart in Myocardial Weakness . . . 118, 119

19. Small Heart in Vasomotor Angina 131

20–21. Myocardial Weakness mainly Involving the Right Ventricle 157

22–24. Cases showing Elevation of the Diaphragm from Collapse of the Left Lung 166, 167

Fig. 22: Normal Chest. Figs. 23, 24: Collapse of Lung.

25–30. Case of Gastric Ulcer, showing a High Diaphragm due to Starvation 170, 171

31–33. Case of Gastric Ulcer showing a High Diaphragm . . 174

34. High Diaphragm the Result of Hæmorrhage (hæmatemesis) 174

35–37. High Diaphragm in Anæmia 175

38. High Diaphragm in Myxœdema 175

39–40. High Diaphragm in Pericarditis 182

41. High Diaphragm in Myocardial Weakness. 183

42–52. Case Showing Variations in the Size of the Liver Dullness, from day to day, due to Heart Weakness . . 196–199

53. Small Heart and Liver Dullness in Arterio-sclerosis . . 202

FIGS. PAGES
54–55. Small Liver Dullness due to Malnutrition 204

56–57. Small Liver Dullness in Neurasthenia 206

58. Diagram showing the Area of Audition of a Systolic Murmur Arising in the Superior Vena Cava . . . 224

59–61. Photographs of Cardiac Ventricles from Above after Removal of the Aorta, Pulmonary Artery, and the Two Auricles 248

Fig. 59: Normal Heart. Fig. 60: Heart with Slightly Dilated Right Ventricle. Fig. 61: Heart from Aortic and Mitral Diseases, with Dilatation and Hypertrophy of both Left and Right Ventricles.

62–63. Lateral View of Distended Hearts 249

Fig. 62: Normal Heart. Fig. 63: Heart with Slight Dilatation of the Right Ventricle Upwards.

64. Semi-diagrammatic View of a Normal Heart as seen in Section through the Pulmonary Artery and Right Ventricle 249

65–66. Case showing Loud Pulmonary Murmur when Recumbent and no Murmur when Erect 256

67–72. Cases showing Pulmonary Murmur and Upward Dilatation of Right Ventricle, both Moderate 260

73–76. Cases showing Loud Pulmonary Murmur with Considerable Dilatation 261

Figs. 73, 74, and 76 with Functional Mitral and Tricuspid Murmurs as well.

77–78. Cases showing an Inconstant and Variable Pulmonary Murmur 268

79–81. Cases showing Moderate Upward Dilatation of Right Ventricle, but no Pulmonary Murmur . . 270, 271

82–85. Cases showing Great Dilatation of Right Ventricle. Pulmonary Murmur Faint 276, 277

86–91. Cases showing Tricuspid and Pulmonary Murmurs both Present 278

92–93. Cases showing Considerable Ventricular Dilatation, but no Regurgitant Murmur 281

94–98. Cases Illustrating the Occurrence of a Right Ventricular Third Sound and Mid-diastolic Murmur . 283, 285, 286

99. Case of Anæmia showing a high Apex Beat and a Normal Diaphragm 288

100. A Case of Anæmia showing a High Apex Beat with a High Diaphragm 289

FIGS. PAGES

101–106. Cases of Anæmia showing Dilatation of the Right Ventricle which mainly Involves the Conus Arteriosus 293, 295

107–108. Two Cases of Anæmia showing Broadening of the Cardiac Area 299

109. Case of Anæmia showing no Increase in the Loudness of the Pulmonary Murmur in the Recumbent Attitude . 302

110–111. Case showing a Pulmonary Systolic Murmur Louder in the Erect than the Recumbent Attitude . . 303

112–113. A Case of Anæmia showing Increased Loudness of the Pulmonary Murmur due to Increased Cardiac Vigour 304

114–115. Case showing Extreme Dilatation and Absence of Pulmonary Murmur 305

116–119. Cases showing Upward Dilatation of the Heart in Overstrain 311, 312, 314

120–122. Case illustrating Dilatation of Right and Left Ventricles in Overstrain in Adult Life 317

123. Low Diaphragm in Case of Overstrained Heart . . 319

124–125. Case showing Extreme Dilatation of Heart from Overstrain in Later Life 321

126. Extreme Dilatation of Heart in Pyæmic Pneumonia . 323

127. Case of Overstrain with High Diaphragm and Small Liver Dullness 326

128. Case showing Downward Displacement due to Intrapericardial Aneurysm 346

129. Case where Rapid Dilatation of Heart with Upward Displacement occurred in Acute Pneumonia . 360

130–132. Cases showing Downward Displacement of the Heart by the Lowness of the Diaphragm resulting from Chronic Over-distension of the Lungs and Thoracic Veins with Blood 364, 366, 367

133. Case illustrating the Normal Percussion of Heart, Liver, and Stomach 369

134. Case showing Displacement Upwards of Heart, Liver, and Stomach 369

135. Case showing Upward Displacement of the Heart by High Diaphragm 370

136. Case showing Rapid Lateral Displacement of the Heart by Collapse of the Left Lung 372

FIGS. PAGES

137. Case showing Displacement of Heart to the Left . . 376

138–140. Case showing Displacement of the Heart to the Right by Gradual Collapse of the Right Lung . . 377, 378

141. Case showing Displacement of the Heart to the Right . 382

142–143. Cardiographic Tracings from a Case of Mitral Stenosis with a well-marked Third Sound . . . 432, 433

Fig. 142 : Tracing taken at Apex, which was in the Fifth Insterpace, 2½ inches from the Sternum. Fig. 143 : Tracing similar to Fig. 142, but taken from near the Apex and when the Heart was acting excitedly.

144–145. Cardiographic Tracings from a Case of Mitral Regurgitation with a well-marked Third Sound . . . 434

Fig. 144 : Record of the First and Second Sounds on a Cardiogram from near the Apex. (Taken in the Fifth Interspace, 2 inches from the Sternum.) Fig. 145 : Tracing taken from the Third Left Interspace, 1½ inches from the Sternum.

146–147. Cardiographic Tracings from a Case of Mitral Regurgitation with a well-marked Third Sound . . . 437

Fig. 146 : From the Sixth Interspace, 4½ inches from the Sternum. Fig. 147 : From the Seventh Interspace 4 inches from the Sternum (i.e. nearly 1 inch internal to the Apex Beat).

148–152. Cardiographic Tracings from a Patient with a very Loud Third Sound, a Faint First Sound, and a very Faint Presystolic Murmur 438, 439

Fig. 148 : Tracing taken at Apex (in the Fifth Interspace, 4 inches from Sternum). Fig. 149 : Tracing taken in the Fourth Interspace, 1 inch from Sternum. Fig. 150 : Tracing taken in the Third Interspace, 2½ inches from Sternum. Fig. 151 : Tracing taken in the Epigastrium. Fig. 152 : Record of the First and Second Sounds on the Cardiogram.

153–154. Cardiographic Tracings from a Case of Mitral Regurgitation and Stenosis with a well-marked Third Sound . 440

Fig. 153 : Tracing from the Apex, showing a Record of the First and Second Sounds on the Cardiogram, and the Theoretical Position of the Third Sound as verified by other Tracings. Fig. 154 : Tracing taken Internal to the Apex where the Diminution in the Volume of the Ventricle at the end of the Systole causes a Fall in the Tracing.

155. Cardiographic Tracing from a Case of Anæmic Dilatation of the Heart with a well-marked Third Sound 441

Tracing from the Apex showing a record of the Second and Third Sounds.

156–163. Tracings illustrating a Case of Arhythmia with Extra Systoles 494–520

Fig. 156 : Tracings from the Apex and the Carotid Artery, showing that the Extra Systole causes no Pulse Wave in the Artery and that the Wave following an Extra Systole is smaller than normal. Fig. 157 : Copy of a Tracing showing a Wave in the Jugular Vein which results from the Extra Systole. Fig. 158 : Copy of a Tracing upon which the Time of Occurrence of the First and Second Sound was marked by an Electrical Signal. Fig. 159 : Tracing Demonstrating the Fact that there is no Sign of any Auricular Wave preceding the Extra Systole.

Figs. 160–163 : Tracings illustrating the Interpretation of the Cardiograms and the Venous Tracings taken in this Case.

PLATES

PLATE

I. A Photograph of the Chest, showing the rapid return of the Heart and Liver Dullness towards Normal after a diminution in size due to Cardiac Weakness . *Facing p.* 64

II. Photograph of the Patient Thos. S., showing the actual Outline of the Heart, Lungs, and Stomach (as determinable by Percussion) marked upon the Chest, for comparison with the Diagrams illustrating the Case *Facing p.* 326

III. Photograph of Thos. S., showing the Outline of the Viscera, as marked on the Chest, as the result of Percussion, antero-lateral view . . . . . *Facing p.* 327

IV. Photograph of the Chest, showing the Outline of the Heart and Liver when Displaced Downwards by the Descent of the Diaphragm owing to Chronic Over-distension of the Thoracic Viscera with Blood . . *Facing p.* 364

V. Photograph of the Chest, showing Downward Displacement of the Diaphragm and Heart owing to the Chronic Over-distension of the Thoracic Viscera with Blood, antero-lateral view . . . . . *Following Plate* IV

VI. Radiograph of the Chest, proving Downward Displacement of the Heart in a Case of Chronic Over-distension of the Thoracic Viscera with Blood . *Following Plate* V

VII. Cardiograms Demonstrating the Enlargement of the Ventricle during its Expansion Phase. (Cases I to III.) *Facing p.* 410

VIII. Cardiograms Demonstrating the Enlargement of the Ventricle during its Expansion Phase. (Cases III. to X.) *Facing p.* 411

TRACINGS ILLUSTRATING A CASE OF ARYTHMIA WITH EXTRA SYSTOLES

(At end of Volume)

IX. No. 1.—Tracing taken Internal to Apex Beat compared with one taken over the Carotid Artery.

No. 2.—Tracing taken at Apex on which was recorded the Time of Occurrence of the First and Second Heart Sounds by means of an Electric Signal.

Nos. 3 and 4.—Upper Tracing, Apex Cardiogram ; Lower Tracing, from Jugular Bulb.

PLATE
X. No. 5.—A Ventricular Cardiogram compared with a Tracing taken simultaneously over the Jugular Bulb.
XA. No. 5A.—Enlarged Photograph of the Original Blue-ink Tracing. part of which is shown in Plate X.
XI. No. 6.—Tracing taken over the Apex compared with one taken over the Jugular Bulb.
No. 7.—Tracing taken a little Internal to the Apex Beat compared with one taken over the Jugular Bulb while the Heart was under the Influence of Digitalis.
No. 8.—Tracing taken a little Internal to the Apex Beat compared with one taken over the Jugular Bulb, while the Heart was strongly under the Influence of Digitalis.
XII. No. 9.—Auricular and Ventricular Tracings compared.
No. 10.—Ventricular Tracing with Record of First and Second Sounds.

HEART FAILURE

PART I.—ESSAYS DEALING MAINLY WITH THE EARLY DIAGNOSIS OF HEART FAILURE

Essay I.—THE EARLY DIAGNOSIS OF HEART FAILURE

By heart failure is meant the inability of the heart properly to discharge its function of circulating the blood.

There is no need to dwell upon the importance of the early diagnosis of heart failure, for the immediate recognition of slight changes in the heart's efficiency may be a matter of vital importance when the medical practitioner is face to face with some serious cardiac complication.

BOSTON MEDICAL LIBRARY
NOV 15

The clinical phenomena which indicate slight alterations in the efficacy of the heart's action do not seem to have received, as yet, the recognition which their importance demands, and therefore much of what will be said in the following pages upon the subject may not receive ready acceptance. When statements such as these are made, however, detailed proof will not be given as a rule at first, although in subsequent essays debatable points will be taken up and clinical proof submitted. Were any other course adopted, the argument advanced would be obscured and overburdened with detail. For instance, in the present essay a simple statement only will be given of the clinical and pathological phenomena which characterise the various types of heart failure, together with the theoretical considerations which harmonise the two sets of phenomena. The full discussion of such signs and symptoms as have not yet received full recognition from the medical profession in general will be deferred to subsequent essays, where clinical proofs can be given of the points raised.

The fact that the earlier phenomena of heart failure are alone to be discussed greatly limits the scope of the subject here dealt with; for although the causes and types of cardiac failure are very numerous, there is nothing like the same diversity in the ways in which cardiac failure of any kind *commences* to manifest itself. Therefore in discussing the early diagnosis of heart failure, it will not be necessary to go into the subject as a whole.

This is due to the fact that, in the majority of the cases, heart failure—whatever its pathological cause—first reveals its presence clinically by phenomena due to a failure of the right ventricle.

There are two reasons for this prominence of the phenomena of right-sided failure :—

Firstly, there are anatomical reasons : in that the right ventricle is more accessible to physical examination than the left, occupying, as it does, most of the anterior aspect of the heart, which is the portion least covered by the lungs. The most dilatable part of the left ventricle, on the other hand, lies deeply in the thorax, and is so completely covered by the lung that dilatation of the left heart cannot be recognised by physical examination until it has reached a very considerable extent.

Another anatomical reason is that the condition of the veins entering the right side can be studied clinically, whereas those entering the left auricle cannot.

The second reason for signs of cardiac failure showing first on the right side of the heart is a physiological one—namely, the well-known fact that when the left ventricle fails, the failure at once throws more work upon the right side of the heart: this being due to the fact that the left auricle is not furnished with a compensation reservoir, as is the case with the right auricle, and therefore there is no means of storing any temporary excess of blood which may result from faulty propulsion on the part of the ventricle. Any delay, therefore, in the passage of blood through the left ventricle must tend to the overfilling of the pulmonary veins and a rise in the blood pressure in the pulmonary artery, and thus throw increased work upon the right ventricle.

Therefore, although the left ventricle is the stronger chamber, it has, owing to the absence of any compensation reservoir, to fall back upon the right ventricle for support in even a temporary embarrassment.[1]

Therefore, since—in the majority of instances—cardiac failure first manifests itself by phenomena due to failure of the right ventricle, the early diagnosis of heart failure consists mainly in the early recognition of an overacting, or a failing, right ventricle.

Before taking up in detail the discussion of the early signs of cardiac failure, it will be well to indicate broadly the lines upon which they will be classified in the following pages.

In bringing under review the various types of heart failure it must be recognised that the type of change which will result from faulty action of the heart will, in any particular case, depend largely upon the degree of elasticity which is possessed by the cardiac tissues. Where a heart is very distensible the symptoms and their interpretation, as well as their treatment, are not the same as where the cardiac tissues are more rigid and possess little distensibility.

This quality of distensibility can therefore be used for purposes of classification, in spite of the fact that all cases of cardiac failure, looked at from this point of view, form a continuous series ranging from the very distensible heart of youth and adolescence, at one end of the series, up to the comparatively rigid heart that is often seen in advanced life, at the other end.

Such a classification, while not of service from a scientific point of view, because of the impossibility of dividing the cases up into well-defined groups by means of it, is yet eminently serviceable from a clinical standpoint. The selection of this characteristic of the heart is amply justified because the clinical phenomena observable in cases at the two extreme limits of the series are very diverse, and the recognition of the amount of distensibility possessed by the heart is so important from the standpoint of prognosis and treatment; also in most cases of heart failure the true meaning of the phenomena presented cannot be understood unless some estimate of the amount of distensibility can be made. Dilatation of a moderately rigid heart shows a far more serious degree of failure than dilatation of an easily distensible heart.

---

[1] This sentence takes for granted the view that the left ventricle is normally complete in itself—filling itself by its own expansion—and is not, under ordinary circumstances, dependent upon the right ventricle at all (*vide* Essay IX, p. 384).

Before going further, this quality of distensibility must be more fully discussed.

When studying clinically and in the post-mortem room, the various types of change in the size of the heart, which characterise cardiac failure, the fact is clearly brought out that considerable diversity is observable, not only in the general distensibility of the organ, but also in the way in which it changes in volume when called upon to try to overcome a hindrance to the circulation somewhat in excess of what it is easily able to meet. In some cases there is evidence that the heart is very distensible and in others that it is not. For instance, on comparing the heart as seen after death with its condition during life, it is sometimes found that a heart which was greatly dilated during life appears after death to show hardly any signs of enlargement; while others will, under the same conditions, show on the post-mortem table almost the same degree of enlargement that was noticeable during life. Again, some patients will die of heart failure without cardiac dilatation under circumstances that *do*, in other cases, produce enlargement. Such facts point to variations in the degree of distensibility of the heart in different patients.

Again, in some cases there is evidence that the thinner parts of the muscular wall are the first to give way, whilst in other patients this is not the case.

If, for instance, we study the way in which the heart dilates in adolescence, we find clear evidence that the main factor which determines the power of resisting dilatation appears to be the thickness of the muscular wall. For the readiness with which any particular part of the heart wall yields to any excess of distensile force—such as occurs in physical overstrain—is found to be proportional to its muscular weakness. The first part to yield is the thinnest part of the right ventricle—namely, that part of the anterior wall which is situated just below the pulmonary valves. The next is the basal portion of the right ventricle, which is intermediate in strength between the muscularly strong apical part and the thin part above mentioned.

Again, the difference in muscularity between the two ventricles seems to render possible very considerable dilatation of the right side in overstrain without any signs that the left ventricle is giving way. Another point in favour of the heart in adolescence having to rely mainly upon its muscular strength for its power of resisting dilatation is that slight degrees of muscular malnutrition seem to play a more important part in favouring dilatation than is the case in later life.

If we now compare the phenomena observable in the adult in cases of overstrain, we shall not find the same readiness of the thinner parts of the heart wall to dilate. The dilatation of the right ventricle is more uniform, and there is not the same disparity in the relative distensibility of the two sides of the heart that was observable earlier in life. In the adult a well-marked dilatation of the right side from overstrain is usually accompanied by recognisable signs of failure of the left ventricle also.

These observations seem to show that while the muscular strength of the heart wall determines its power of resisting dilatation in early life, in later life dilatation is prevented by some other factor. This factor is the fibrous tissue which enters into the structure of the heart and pericardium.

Some work done by the late Dr. Arthur Foxwell clearly showed that the fibrous tissue of the pericardium, and especially that of the parietal pericardium, had an important function in preventing dilatation of the heart.

The recognition of this fact makes the solution of our problem an easy one, for, in the rapidly developing tissues of adolescence, the resisting power of the fibrous elements is very low as compared with their strength later in life.

We can therefore make the following generalisations :—

In youth and adolescence the heart, owing to the softness and elasticity of the fibrous tissues which enter into its composition, has mainly to rely upon its muscular tissues for its power of resisting dilatation. In middle life, when the fibrous tissues become firmer and less distensible, they take a much larger share in the prevention of over-distension than they do in earlier life.

In later life the relative rigidity and non-distensibility which sometimes characterise the heart are presumably due to an extreme hardening of the fibrous elements in its composition.

Enough has now been said to explain what is meant by distensibility of the heart as a basis for classification, and the further discussion of this quality can be postponed until after the subject of Classification has been dealt with.

CLINICAL CLASSIFICATION OF CASES OF CARDIAC FAILURE

From the point of view of early diagnosis, all cases of cardiac failure must be divided into two main classes; for while the *symptoms* of early heart failure may often be very similar, a marked difference is recognisable in the *physical signs*; and all cases, whether in distensible or in more rigid hearts, must be divided into two main classes—namely, (1) heart failure with enlargement of the heart, and (2) heart failure without enlargement.

In the first group the heart when having to overcome a resistance to the outflow of blood, which is in excess of the resisting power of its walls, is able to raise its intraventricular pressure sufficiently to empty itself; but in so doing causes over-stretching of its walls. (Whatever be the theory as to the mode in which dilatation is produced, the ultimate force producing it *must* be the muscular power of the heart.)

In the first group, therefore, the heart when called upon to do work beyond the resisting power of the weaker parts of its walls is strong enough to do so, and, consequently, dilatation results. This group, where the heart muscle is relatively strong, may be called *Cardiac Failure with Enlargement of the Heart.*

In the second group are included cases where the heart muscle is too feeble to cope with the resistance which it has to face. It cannot raise the intraventricular pressure to a point sufficiently high to produce dilatation of the heart. In this group we have to deal with *cardiac failure without enlargement*, and as a well-known illustration may be cited the case of fatty degeneration, where a patient can die of heart failure without showing any signs of dilatation, of engorgement of the veins, heart murmur, or other of the generally accepted physical signs of heart failure.

For the purpose, therefore, of discussing clinically the early diagnosis of heart failure, it is proposed to subdivide the subject as follows:—

1. Heart failure with enlargement, as it occurs in—
    - (*a*) The distensible heart of adolescence;
    - (*b*) The moderately distensible heart of middle life;
    - (*c*) The more rigid heart of advanced life.
2. Heart failure without enlargement, as it occurs in—
    - (*a*) The distensible heart of adolescence;
    - (*b*) The moderately distensible heart of middle life;
    - (*c*) The more rigid heart of advanced life.

DISTENSIBILITY OF THE HEART IN ADOLESCENCE

Before taking up the discussion of the first of these groups it will be well to go a little more fully into the quality of distensibility as manifested in adolescence.

AMOUNT OF DISTENSIBILITY.—In early life the amount of cardiac distensibility is sometimes remarkably great, and the following is the most striking instance of it that I have met with.

The patient was a young woman, sixteen years of age, who was suffering from pyæmic pneumonia. The lung disease was most extensive, and at the post-mortem the lungs were found to be full of pyæmic abscesses. When I saw her, there was an enormous amount of dilatation of the heart. The cardiac dullness and pulsation extended from a point one inch to the right of the sternum to beyond the left anterior axillary line and from the first left interspace above down to the fifth interspace in the anterior axillary line. There were the usual murmurs present. The patient died two or three days later. On inquiring as to the condition of the heart from the pathologist who made the post-mortem examination, I was surprised to hear that it was of normal size. Fortunately, I was able to examine the organ for myself and found that there was no evident enlargement. On testing its walls, however—and especially those of the right ventricle and the auricles—they were found to yield to an amount of force that would not have been sufficient to stretch normal heart walls. With such a degree of distensibility it was easy to believe that the heart walls would readily yield to a raised blood pressure, and that, therefore, its smallness post-mortem was *not* incompatible with dilatation during life (see also p. 322).

Here, then, was a case where the heart was distensible to a most remarkable extent without exceeding the limit of its elasticity, and so was able to return to its normal size as soon as the distending force was removed by death and its contractility increased by rigor mortis.

The ready distensibility of the heart in adolescence is also very striking so far as the thin upper part of the anterior wall of the right ventricle is concerned, and such an observation as the following may easily be made. A young woman of seventeen or eighteen, who is suffering from simple anæmia with breathlessness, is being examined in the out-patient department of a hospital, and it is found that there is a slight amount of cardiac dullness and pulsation in the second left interspace suggestive of anæmic dilatation of the conus arteriosus of the right ventricle. After the extent of the dilatation has been carefully noted several students are called into the room to examine the patient, and it will then not infrequently be found that the nervous excitement caused by their presence will, for a time, greatly increase the amount of the dilatation. Or, to take another instance, a young man has by over-indulgence in athletics caused an over-stretching of the same thin portion of the right ventricle. If such a patient be examined immediately after exertion, a much greater amount of dilatation of the heart in the second left interspace may sometimes be observable than would be found after a few minutes' rest.

Such observations as these show that in youth and adolescence the heart is not only readily distensible, but also possesses a high degree of elasticity, admitting of a rapid return to its former size as soon as any abnormal distensile strain has lessened.

This quality of elasticity is characteristic of adolescence, and is, as a rule, less and less noticeable as the patient's age increases.

TYPE OF DISTENSIBILITY IN ADOLESCENCE.—The heart in adolescence shows special features as regards the different degree in which the various parts of its walls yield when there is any excess of internal pressure. It was pointed out at p. 4. that these special features were due to the fact that the distensibility of any particular part of the heart wall appeared to be proportional to its muscular weakness as compared with the rest of the heart wall.

In connection with the present subject it is necessary again to refer to the comparative muscular strength of the various portions of the heart wall.

In the first place the right ventricle is evidently weaker than the left, and the relative strength of the various parts of its walls are as follows:—

The thinnest part, and therefore the weakest muscularly, is the portion of the anterior wall which lies nearest to the pulmonary artery, and includes the conus arteriosus.

The next in order of muscular strength is the portion of the anterior and right lateral walls which is adjacent to the auriculo-ventricular orifice.

The strongest portions are the apical part with its strong muscular bands, and the interventricular septum.

As regards the left ventricle, the basal part of its wall which is adjacent to the auriculo-ventricular orifice is relatively weaker than the apical.

As the strength of a chain is that of its weakest link, if the ventricle depended upon its muscular wall alone for its power of resisting dilatation due to an abnormal rise in the intraventricular pressure, the first part of its wall to give way would be the thinnest portion.

Now, supposing that the resistance to be overcome in the pulmonary artery is raised to such a degree that it exceeds the resisting power of the weakest part of the muscular wall of the right ventricle but does not exceed the strength of the stronger portions, the intraventricular pressure will, when it reaches its maximum, cause a yielding and stretching of the weakest part above named.

We can express this in general terms by saying that, in such hearts as we are speaking of, when the resistance to be overcome in the pulmonary artery exceeds the resisting power of the weakest part of the ventricle wall, but does not exceed the strength of the strongest part, then the weaker parts of the wall are over-distended by the action of the stronger, thus causing dilatation.

In connection with this special distensibility of the upper part of the anterior wall it must be borne in mind that the final expulsive efforts of the ventricle are concentrated on this part of the wall which lies nearest to the outlet; for in spite of the physical law that in any cavity containing liquid the pressure is equal in all directions, the strain must be more prolonged on this—the last portion of the ventricle to be emptied.

What has been said may be summed up as follows:—

In adolescence, when, owing to the softness and immaturity of the fibrous tissues the heart cannot rely on them for support, it requires but a slight increase in the amount of work the heart has to do to cause some dilatation of the conus arteriosus of the right ventricle, more especially if the heart muscle itself be lacking in tone from anæmia or other cause.

When the fibrous tissues become less elastic, in middle and later life, the right ventricle not only loses its ready dilatability, but its type of dilatation changes also; for the conus arteriosus no longer shows the special weakness that it does in earlier life, and, under these circumstances, we find that the first part of the ventricle to yield, when over-strained, is the basal portion of the anterior and right lateral wall in general, and not the small portion below the pulmonary valves.

This increase in the strength of the wall of the right ventricle in middle and later life is naturally associated with a relatively increased frequency in the occurrence of dilatation of the left ventricle, as the result of overstrain.

In adolescence, when the heart is overstrained, early failure of the weak right ventricle saves the left from dilatation. In adult life it is usual to find the left ventricle at least as much dilated as the right, and when the fibrous tissues have become relatively rigid, we may find dilatation of the left side more extensive than that of the right. This will be referred to again, later on, in speaking of failure of the left ventricle.

The special distensibility of the conus arteriosus, therefore, is found to diminish as the fibrous strength of the heart and pericardium increase with advancing years; and although the period of life when the change takes place varies very much in different individuals it is fairly safe to say that the special dilatability of the conus arteriosus is lost as a rule towards the end of the third decade; and in patients over thirty-five it is not common to find failure of the right ventricle, from overstrain or other cause, accompanied by the dilatation upwards and to the left which characterises a yielding of the conus arteriosus. In such patients the dilatation will most commonly be a general one of the base of the right ventricle, and the enlargement of the heart will be to the right rather than upwards and to the left. This, which may well be called the adult type of dilatation, may sometimes occur in patients much younger than the age above mentioned, and, on the other hand, the adolescent type may sometimes be found in patients well over fifty years of age.

Certain conditions of ill-health, however, appear to be accompanied by a tendency to softening of the fibrous elements of the heart walls, thus increasing their distensibility: this is notably the case with rheumatic fever. After a severe attack of rheumatism, when the patient first begins to get up, the conus arteriosus will often be found to dilate far more readily on slight exertion than would be the case after such an illness as pneumonia. It is possible that this softening effect of rheumatism is of value in allowing the necessary compensatory dilatation to take place in mitral and aortic regurgitation, and is therefore to that extent beneficial. When a case of aortic or mitral regurgitation occurs in a patient whose heart walls are too rigid to admit of compensatory dilatation, it shows what a serious thing such valvular disease would prove to be, were it not for the ready distensibility of the heart walls in most of the patients who suffer from rheumatic endocarditis (*vide* Essay on Pallor, p. 448).

After an attack of rheumatism, or some allied ailment, it may be found that in a patient over thirty the conus arteriosus seems to be as dilatable as in a normal patient of twenty or twenty-five years of age.

EARLY DIAGNOSIS OF HEART FAILURE WITH ENLARGEMENT IN DISTENSIBLE HEARTS

After what has just been said as to the relative strength of the right and left sides of the heart in early life, it will be recognised that the early diagnosis of heart failure with enlargement in early life is simply the early diagnosis of dilatation of the right ventricle. It has already been pointed out that dilatation of the right ventricle in early life shows itself first as an upward and outward dilatation of the part of the anterior wall which lies just below the pulmonary valves, and therefore it is to the symptoms and physical signs of this type of dilatation that we must look for the earliest indication of failure in a distensible heart.

SYMPTOMS.—The early signs and symptoms, which are characteristic of this condition, will first be discussed, and then subsequently their relative order of occurrence and their diagnostic importance.

DYSPNŒA is in this, as in all cases of cardiac failure, one of the earliest indications that the heart is unequal to the work it has to do.

The breathlessness of right ventricular failure in adolescence has, however, some special characteristics.

In the first place its amount is often very great, especially when associated with anæmia, as is so commonly the case.

When right ventricular failure accompanies a severe anæmia, the breathlessness may be so great that the patient cannot go up more than three or four steps without stopping to rest.

Such an amount of dyspnœa under other circumstances would mean serious organic disease of the heart, but in the case of dilatation of the right ventricle in an anæmic adolescent a few weeks of rest, and the administration of iron, will often suffice to restore the working power of the heart.

A special characteristic of the breathlessness associated with this adolescent type of heart failure is the well-known tendency to take deep sighing breaths. These respirations consist of a full inspiration which is held as in an ordinary sigh, although for a slightly longer time, and then the air in the chest is suddenly allowed to escape.

This type of respiration gives distinct relief in this kind of case.

The physiological basis for this fact may for the sake of completeness be referred to here.

When the right ventricle is somewhat over-burdened and has a difficulty in expelling its contents, owing to the abnormal resistance in the pulmonary artery, the long and powerful inspiration has the effect of drawing an extra amount of blood into the great veins and the right heart. The breath is presumably held until such time as the commencement of expiration coincides with the contraction of the ventricle. As a result of this, the expiratory increase in the intra-thoracic pressure comes at the right time to give some measure of support to the weak and stretching walls of the right ventricle, thus aiding its expulsive power and also, by increasing the external pressure upon the veins and auricle, lessening the tendency to tricuspid leakage, if there be any, and helping the subsequent filling of the ventricle.

Clinical proof can easily be obtained that the expiratory increase of intrathoracic pressure does materially aid the heart's action in this type of heart failure.

Thus in cases where the pulsation of the right ventricle is visible in the third and second left interspaces (p. 18), it can be seen that the first systole after the commencement of expiration causes a larger wave of pulsation than other beats.

Also where accurate means are adopted (*vide* p. 413) of measuring the loudness of the heart sounds it is often noticeable that the first pulmonary second sound to occur after the end of inspiration is louder than the others. Further, in the case of murmurs produced by the action of the right ventricle it is often noticeable that they are louder at the commencement of expiration than during the remainder of the respiratory cycle.

This taking of a long breath is common in all cases of right ventricular failure in distensible hearts, but especially so where there is anæmia as well.

The relief it gives is so well known to the patients themselves that, when the heart gets worse and the same relief is no longer obtainable, they will sometimes answer the doctor's question 'What do you complain of?' by saying 'I can't take the long breath.'

Before speaking of the physical signs observable in this type of heart failure it will be well to say a word or two as to certain other symptoms of heart failure from which patients suffer.

PAIN.—Occasionally a certain amount of discomfort or dull pain can be felt over the situation of the dilated conus arteriosus, and in some patients this sense of discomfort precedes the occurrence of breathlessness and tells the patient that the exertion which is being taken is overtaxing the heart.

GLOBUS HYSTERICUS.—A less common symptom is that of a lump in the throat, as if too large a bolus of food had been swallowed and was lodged in the lower end of the œsophagus. This sensation is possibly due to the pressure of an over-distended left auricle upon the œsophagus in a sensitive patient. I have only met with it in the distensible hearts of adolescence, but it may occur in other conditions.

CLINICAL PHENOMENA ASSOCIATED WITH THE VEINS.—PULSATION in the veins of the neck.—One of the earliest symptoms of this, as of other types of right ventricular failure, is the undue fullness of the veins of the neck, which results from the difficulty which the right ventricle experiences in forwarding the blood through the lungs at the normal rate.

At first a simple fullness of the veins is observable. Later, when the delay is greater, a distinct variation in their fullness can be detected, and the character of the variation is dependent both upon respiratory and upon cardiac movements.

The respiratory variation consists in an increased fullness during expiration and a relative emptying of the veins during inspiration.

This respiratory rhythm is superimposed upon the variation due to the heart. This double variation is known as FALSE PULSATION in the neck veins.

The cardiac variation in early cases of failure consists in what is apparently a systolic filling of the veins. On closer examination, however, it can easily be recognised as the type of pulsation which is not a true but a false venous pulsation. In this type, the variation consists in a rhythmical emptying of veins that are unduly distended and not in a rhythmical filling of veins that are relatively empty—such as occurs in tricuspid regurgitation. Proof of this is given by the fact that the pulsation is apt to be more marked and distinct during inspiration than during expiration—that is to say, the amount of cardiac variation in the fullness of the veins is increased by anything aiding the *entrance* of blood into the heart. In true tricuspid regurgitation the reverse condition is found, for the pulsation, where it varies at all with respiration, is always more distinct during expiration than during inspiration; for the escape of blood from the heart into the veins is favoured during expiration, whereas inspiration tends to hinder it. Associated with this false pulsation, which characterises cases of right ventricular failure where there is no marked tricuspid regurgitation, we find, under certain conditions, a definite venous hum or murmur, most marked in anæmic states, and commonly known as the—

‘BRUIT DE DIABLE.’

This murmur, as is well known, is due to the fact that when the neck veins are over-distended there are certain points where they receive enough external support from the fascia, &c., to prevent them from dilating, and thus constrictions are formed where murmurs can arise. If the veins be over-distended with hard paraffin, it will be found that a constriction occurs wherever a small vein joins a larger one, owing to the support received by the small vein at its point of entrance into the wall of the larger one. The orifice of entrance, therefore, of the smaller vein does not dilate to the same extent as the vein itself does.

This murmur, which is low-toned and humming, is found to vary in tone with both the respiratory and the cardiac rhythm, and in moderately severe cases it disappears entirely when the patient lies down. This is due probably to the fact that in the recumbent attitude the veins in the clavicular region are less distended, owing to the elimination of the action of gravity. In confirmation of this it may be noticed that on lying down the veins in the upper part of the neck become more distended than they are when the patient is erect. This variation in the fullness of the veins is specially noticeable where there is the poor venous and cardiac tone which characterises cases showing a ' bruit de diable.'

True Pulsation in the neck veins.—The true systolic venous pulsation, which characterises tricuspid regurgitation, is easily distinguished from this false pulsation with which we have just dealt. True venous pulsation does not, as a rule, occur with the type of cardiac failure of which we are now speaking.

The dilatation of the conus arteriosus is due, as already pointed out, to a rise in the systolic ventricular pressure beyond the point that the muscular walls can stand. Such a rise in the interventricular pressure implies either a competent tricuspid valve or else a degree of auricular and venous tonic contraction sufficient so far to limit the tricuspid regurgitation as to make possible a rise in the intraventricular pressure above its normal level. In adolescence, however, such strength in the musculature of the veins and auricle is not to be expected, and therefore we do not expect to find tricuspid regurgitation associated with upward dilatation of the ventricle as an early sign of heart failure in distensible hearts.

In severe cases, however (especially in those where the failure of the right ventricle is secondary to valvular disease of the left), marked dilatation upwards may, even in distensible hearts, be accompanied by some general dilatation of the right ventricle, auricle, and veins such as characterises heart failure in adult and later life.

The subject of true systolic pulsation in the veins of the neck may therefore be deferred until cardiac failure in less distensible hearts is dealt with.

Physical Signs.—The chief physical signs upon which we have to rely for the early recognition of heart failure in distensible hearts are due to the special direction in which such hearts dilate—namely, upwards and to the left, in consequence of the yielding of the thin part of the right ventricle.

Inspection of the Heart.—One of the first signs of failure of a distensible right ventricle is the appearance of pulsation at the sternal end of the fourth left interspace, or its increase in amount if it have been previously present there.

With increasing dilatation, pulsation will appear in the third left interspace, and, subsequently, in the second, if the right ventricular dilatation be considerable. In such a case there will be visible pulsation in the second, third, and fourth interspaces, and it may extend from the left edge of the sternum to the vertical nipple line in the second left space, and may sometimes reach to the anterior axillary line in the third and fourth. In such a marked case there will be so much of the right ventricle uncovered by lung tissue that the character of the pulsation will be clearly distinguishable.

The presence of this pulsation was first described, I believe, by the late Dr. Foxwell in his Cambridge graduation thesis, and subsequently in his 'Bradshaw Lecture' and his 'Essays in Heart and Lung Disease.'

Its features are quite characteristic. It has a fluttering appearance, and is not the simple rise and fall such as is seen, for instance, in aneurysm. This irregularity is due to its variation with respiration, and also to the fact that it has a sort of wave-like motion: the line of maximal rise travelling from one side of the pulsating area to the other like an ordinary wave-crest on water.

The respiratory variation consists in a greater degree of visible movement during expiration than during inspiration. This will at first be thought to be due to the heart being less covered by the lungs during their expiratory recession; but on making the patient hold the breath with the lungs fully emptied it is found that the pulsation is less than it is during the continuance of ordinary respiration.

If the pulsation be still more carefully watched it can usually be noticed that the maximal degree of pulsation occurs with the first cardiac systole which follows the end of inspiration, and sometimes the systolic impulse with this beat is decidedly greater than that due to the other heart beats.

The other characteristic of this right ventricular pulsation is the visible passage of the wave of contraction across the pulsating area.

Considering that the blood is being emptied out of the ventricle through an orifice (the pulmonary artery) which lies under the second rib and some one inch or more to the left of the sternum, it might be expected that the peristaltic wave would be seen passing up from the apex and gaining in volume and distinctness as it approached the pulmonary artery—as is the case with the muscular wave which causes the emptying of the stomach.

The direction of the muscular movement in the right ventricle has, however, quite a different direction from this. It first appears travelling out from under the sternum in the second or third interspace and travels downwards and outwards over the exposed part of the right ventricle more or less parallel to the line of the auriculo-ventricular groove, though inclined rather more downwards, and also more or less parallel to the direction of the pulmonary artery.

The line of crest runs in a straight not a curved line, and its direction of movement is a line inclined some thirty degrees to the vertical, whereas that of the auriculo-ventricular septum would be more nearly vertical, say, with an inclination of some twenty degrees or less. The direction of this wave seems compatible with modern myogenic theory as to the origin of the stimulus to contraction.

The other points, which may be gathered by inspection of the cardiac area, are the absence of pulsation to the right of the sternum, owing to the limitation of the dilatation to the portion of the anterior wall near the pulmonary valves and also the abnormal position of the apex beat, which will be further referred to when dealing with palpation, and which is also fully discussed at p. 348.

Palpation.—Study of the cardiac impulse by palpation confirms the supposition (based on its respiratory variations and other characteristics) that it is due to the right ventricle. It is not a powerful pulsation like that of the left ventricle, and is better studied by inspection than by palpation.

Palpation is, however, of great value as a quick and simple means of detecting the presence of this type of dilatation of the right ventricle; for the occurrence of true cardiac pulsation in or external to the nipple line in the third interspace is (allowing for the uncertainties common to all medical statements) pathognomonic of this condition. The presence or absence, therefore, of upward dilatation can, when well marked, be easily determined by careful palpation, with the tip of one finger, in this situation. If pulsation be felt, dilatation of the conus arteriosus of the right ventricle can be diagnosed, and if there be no pulsation in this situation it is almost equally certain that there is no dilatation.

Before making this diagnosis, however, the absence of marked displacement of the heart from disease of one or other lung must be excluded.

Another fact to be looked for by palpation is the altered position of the apex beat. Often in these cases the cardiac apex is higher than normal, being found in the fourth interspace instead of the fifth, and is also external to its proper situation. In well-marked cases the heart's apex may be in the fourth interspace, well outside the vertical nipple line, and sometimes not far from the anterior axillary line.

The causes at work producing this change are discussed fully at pp. 234 and 348, to which the reader is referred.

The presence of a palpable thrill over the pulmonary artery may be detected in severe cases of this type, but more especially where upward dilatation of the right ventricle is associated with anæmia.

Percussion of the Heart in adolescent dilatation of the right ventricle.—Percussion is as valuable as palpation in determining the presence or absence of this type of dilatation, but does not take the premier place as a diagnostic method because, as a rule, the cardiac vigour which is necessary for the development of dilatation will also ensure the presence of an amount of pulsation which can be recognised by palpation.

By percussion, however, the exact size and shape of the cardiac area can be determined in greater detail than by palpation.

Before, however, going further with this subject it will be well to say a few words upon the question of percussion in general—more especially because a good deal of the clinical evidence advanced in support of the theories here propounded is based on the results of percussion.

The Principles and Method of Percussion

First as to method. The finger as pleximeter has many advantages over any artificial substitute, and not the least of these is the information gained by the resistance offered by a solid or partially solid organ underlying it, as compared with an air-containing one. So much is this the case that it is possible for the inexperienced percusser, when suffering from the usual periosteal irritation which results from too much enthusiasm in the newly acquired art, to tell by the varying degrees of pain caused by the percussion stroke the degree of density of the organ percussed.

The plessor, too, had better be the finger of the clinician rather than a hammer of any sort, for the finger is capable of a far quicker blow than any hammer, and the clearness of the percussion note is dependent upon the quickness of the blow whatever its force may be. A sustained blow cannot fail to check the fullness of the vibration to which the percussion stroke gives rise.

As to the most useful of the two kinds of percussion, there is no need to raise the old controversy as to which is the best mode—whether gentle or strong; for the use of both kinds gives us such valuable information that we cannot afford to discard either. We have need to observe both the area where the dullness of a solid organ is complete, as well as the area where that dullness is modified by the presence above or below it of a resonant air-containing viscus.

As to terminology, it would be an advantage if the terms 'absolute' and 'relative' dullness, or some equally unmistakable ones, were generally adopted instead of the more equivocal terms 'superficial' and 'deep' dullness. Some medical writers seem to use these latter terms in opposite senses, and it certainly is possible to take the term 'deep' dullness to apply to the fully dull note of a solid organ at the surface, as well as applying to the note of a dull organ lying deeply. Another disadvantage in the use of the term superficial dullness is that a solid organ, although superficial, may give only a relatively dull note (' deep dullness ') if underlaid by a resonant organ. In the following pages the terms 'absolute' and 'relative' will be used, and they have the advantage of being also serviceable in describing the resonance of such an organ as the stomach or colon, the absolute resonance being the full resonance obtainable when the percussion note is not damped by any superimposed solid organ; and the relative resonance being the partial resonance obtainable when the liver, or other intervening solid tissue, modifies the amplitude of the note.

In the illustrations which show the results of percussion, the absolute dullness will be marked by dark shading, and the relative dullness, whether due to the presence of an air-containing organ overlying or underlying the dull one, will be marked by lighter shading.

As regards the information gained by percussion.

Firstly, take the case of a dull organ which is overlaid in part by an air-containing one, such, for instance, as the upper border of the liver and the upper and outer border of the normal heart, where they come into relationship with the lungs. Here, by very light percussion, we endeavour to map out the exact edge of the lung, calling the area uncovered by lung the absolute dullness of the liver or heart respectively. In the same way, by stronger percussion, we endeavour to define the area over which some damping of the full lung note can be recognised (owing to the presence of the underlying solid organ), and we call this area the relative dullness of the liver or heart respectively: though we should be equally accurate in calling it the area of relative lung resonance. The width to which this area of relative dullness is found to extend around the area of absolute dullness naturally depends to some extent upon the skill of the observer and the depth of lung tissue which he is able to cause to vibrate as the result of his percussion stroke. The less effective stroke failing to reveal the presence of the dull organ at so great a depth as the more effective one.

This variation in the boundary of the relative dullness, according to the skill displayed, has been advanced as an argument against the clinical value of recording the relative dullness. But the variation due to this personal equation is not great enough to give much force to this argument.

The movement of the edge of the lung during respiration must naturally be taken into consideration, and for very exact records it is necessary that the breath be held in the middle of expiration or inspiration. In the figures given in the following pages the line is the average level—unless otherwise stated.

In determining the area of the relative dullness it must not be forgotten that mobility of the lungs is of value in confirming the line which is decided upon as marking the boundary of the relative dullness. By percussing again, when the patient has fully emptied the lungs, the correctness or otherwise of the previous observation can easily be verified.

It has previously been stated that the observation of both the absolute and the relative dullness of an organ gives valuable information. Let the upper level of the liver anteriorly be taken as an example. To note the situation of the absolute or of the relative dullness alone gives simply the approximate position of the upper level of the liver. If, however, both are noted, it is possible to estimate the rate at which the liver is receding from the surface—or, in other words, the rapidity with which the lower edge of the lung thickens. The narrower the band of relative dullness which intervenes between the full resonance of the lung and the full dullness of the liver the more sudden must be the recession of the diaphragm and liver from the chest wall, and the broader the relative dullness the more gradual that recession.

In this way it is often possible to estimate the degree of arching of the diaphragm. If the breadth of the area of relative dullness exceeds the normal it suggests that the diaphragm is abnormally arched. If less than the normal, that it is either unduly flat or else that the edge of the lung is distended in the manner that occurs in emphysema.

The broad zone of relative dullness which suggests abnormal arching of the diaphragm is well shown in fig. 31, p. 174.

Secondly, where a dull organ is underlaid by a resonant one the method of procedure must be reversed, so far as the area of the dullness of the organ is concerned; for its area of absolute dullness must be determined by strong percussion, and the boundary of its area of relative dullness by light percussion. This is best illustrated by the percussion of the lower edge of the liver, where it overlies the resonant stomach or intestines. It requires a gentle stroke to define the thin edge of the liver, where it lies over the resonant stomach, and a stronger blow to elicit the resonance of the stomach or bowel when it underlies some thickness of liver tissue. To define the absolute dullness here requires therefore a strong stroke, and to define the relative dullness a gentle one. Information of clinical importance can be obtained by noting the lower boundary of the liver dullness through a series of cases; for it shows great variation, and in cases of defective circulation there may be very great diminution in the area usually occupied by the dullness of the lower part of the liver. This subject is discussed in the Essay on Diminution of the Liver Dullness, at p. 185.

These two methods of obtaining the absolute dullness of the liver may be harmonised and embraced by a single generalisation as follows:—

In defining by percussion the boundary between an organ giving a dull percussion note and one giving rise to a resonant note, the boundary-line along which the underlying one comes to the surface must be determined by light percussion, and the farthest point where its note can be detected through the overlying one by strong percussion. In other words, the absolute dullness or absolute resonance of the underlying organ must be obtained by light percussion, and its relative dullness or resonance by strong percussion—e.g., the lower edge of the liver when marking the limit of full intestinal or gastric resonance needs light percussion, the lower border of the absolute hepatic dullness, marking the upper limit of the relative or partial gastric (or intestinal) resonance, needs strong percussion.

A further generalisation is also in the main accurate—namely, that the rate at which the underlying organ is receding from the surface may be inferred from the width of the area which lies between these two boundary-lines—i.e., of the area of relative dullness. The nearer these lines are together the steeper the angle at which it is receding, and the farther they are apart, the less the angle, and the more gradual the recession.

While speaking on the subject of percussion it may be well to draw attention to a further point, which will be of importance in a subsequent essay (see p. 191), although it does not concern the present one. This point is that this last-named generalisation does not always seem to hold good so far as the lower edge of the liver is concerned, for under certain circumstances a diminution in the amount of liver dullness in the right hypochondrium does not always seem to be due to a lessening of the amount of liver substance that intervenes between the point percussed and the resonant intestines underneath. Sometimes the edge of the liver can be clearly felt at, or below, its normal situation when the area over which the full intestinal resonance is obtainable on percussion extends far above its normal limits; thus suggesting that the intestinal resonance must in this case be obtainable through a far thicker mass of liver tissue than is normally possible.

There is, I think, no doubt that under certain pathological conditions the liver tissue loses its power of damping down percussion vibrations.

The subject is, however, one needing further research, and it will probably be found that the possession of this quality is a sign that the liver tissue is unduly tense through congestion (or unduly hard), and, therefore, has the power, when distended bowel is in close contact with it, of conducting the vibrations of the percussion stroke to the bowel, and the vibrations of the bowel wall back to the surface.

It is quite probable that the possession by the liver of this increased power of conducting percussion vibrations may prove, when fully investigated, as valuable a sign of congestion or hardening of the liver substance as the power of conducting aerial vibrations has proved to be in the case of consolidation of the lung (see also pp. 189–193).

The occurrence of such unexpected percussion results as this in the case of the liver may have discouraged clinicians from studying it, and helps to account for the neglect which has been so marked in the past in connection with this branch of clinical observation.

PERCUSSION of the heart in the adolescent type of dilatation of the right ventricle.—The increase in the size of the right ventricle upwards and to the left, which characterises this type of dilatation, will naturally lead to an increase in the amount of cardiac dullness in this direction—namely, an increase in the normal amount of dullness in the third interspace and the appearance of abnormal cardiac dullness, first relative and later absolute, in the second interspace. In extreme cases there may be absolute cardiac dullness for 2 inches or more in the second left interspace and to a corresponding extent in the third, and the relative dullness may even reach up into the first interspace. Increase of the cardiac dullness of this type is not as a rule accompanied by any increase to the right, and in these cases the sternum is usually resonant down to the level of the fourth interspace or fifth rib. This resonance of the sternum does not mean that the heart is not in close contact with it till such a low level is reached, for the sternum being a single flat bone takes the note of that structure with which it is mostly in contact. If the lung be in contact with its under-surface for more than half its length the note it gives is mainly resonant; if the heart, on the other hand, be so enlarged as to displace the lung for more than half its length the sternum will be mainly dull. This fact accounts for the sudden changes in the size of the area of the cardiac dullness which may sometimes be noted when a heart which has been occupying rather less than half the length of the sternum increases a little in size so as to occupy rather more than half, or vice versa.

The absence of dilatation to the right of the sternum has already been referred to, and is well shown in fig. 99, p. 288.

In well-marked cases of dilatation of the conus arteriosus the outline of the heart varies considerably, and its general characteristics are sufficiently well shown in figs. 68–75, and do not call for further description here, where we are dealing with early diagnosis only. The fact of an increase in the amount of dullness in the recumbent position as compared with the amount when the patient is erect is also dwelt upon, and the reasons for this change, given in the Essay on the Condition of the Heart in Anæmia.

Auscultation of the Heart in Adolescent Dilatation of the Right Ventricle.—Auscultation cannot always be relied upon as a means of diagnosing adolescent dilatation of the right ventricle, because auscultatory signs may be absent even in well-marked cases; they are as a rule, however, both definite and characteristic.

The chief sign of this upward dilatation is the occurrence of a systolic murmur in the pulmonary artery. The clinical and pathological discussion of this murmur will be found at pp. 247–253 and p. 254, and it is there shown that the features of this murmur are as follows:—

It is produced in the pulmonary artery under the following circumstances. Owing to the upward extension of the anterior wall of the right ventricle the level of the pulmonary valves is raised, and therefore the point of origin of the pulmonary artery is brought nearer to its termination, which is a fixed point. There must, therefore, be some degree of relaxation of its elastic walls, and when the blood pressure rises during systole this relaxation will result in the development of an aneurysm-like dilatation of the artery. In this a murmur is produced just as is the case in an ordinary aneurysm. The main factors, therefore, which are concerned in the production of the murmur are: firstly, the relaxation of the elastic wall of the pulmonary artery; secondly, a blood pressure adequate for its over-distension; and thirdly, a pulmonary orifice sufficiently small in comparison with the dilated artery to admit of the formation of murmur-producing eddies in it.

The greater the relaxation the louder therefore will be the murmur, if the other factors are equal. Where there is more lateral dilatation of the ventricle and less upward dilatation a less loud murmur will be heard than where the dilatation is mainly upward. Also, in the erect posture the weight of the heart tightens the artery more than is the case when the patient is recumbent, therefore the murmur is, as a rule, less loud in the erect position.

From what has just been said it will be seen that the essential point in the production of the murmur is the distension of the artery so that the diameter of its lumen exceeds that of the pulmonary orifice. Were the pulmonary orifice itself to be dilated to nearly the same extent as the artery, no murmur would be produced by the inrush of blood into the dilated artery. In many cases of extreme upward dilatation—when, for instance, the pulmonary valves are at the level of the upper border of the first rib or higher—it is found that a pulmonary murmur is not present, or if present is only very faint, and the reason is almost certainly that just stated—namely, that the pulmonary orifice is dilated as well as the pulmonary artery.

Also, for the production of the murmur there must be sufficient distending force. The amount of distension will be proportional to the excess of the internal pressure in the artery over the external resistance. Therefore, when the heart is weak a comparative feebleness, or even an absence of murmur, may be observable in spite of considerable dilatation of the ventricle. In such a case the murmur will be found to appear as the heart gains in strength (*vide* p. 305). To take the opposite extreme, a very loud murmur usually occurs in anæmia because there is, in this condition, a marked rise in the blood pressure in the pulmonary artery (as shown by the loud second sound, *vide* p. 244) as well as upward dilatation of the ventricle.

It is possible also that increased support to the artery, from consolidation of the adjacent lung or other cause, would diminish the loudness of the murmur by limiting the amount of dilatation.

To sum up: a loud murmur is to be expected in cases of absolute overstrain unless the pulmonary orifice be dilated as well as the artery, or unless there be sufficient resistance from adjacent structures to prevent the over-distension of the artery; and a faint or absent murmur is to be expected in cases of relative overstrain where the ventricular dilatation, is due to weakened ventricular walls, coupled as this must be with a lowered blood pressure in the pulmonary artery as a result of the muscular weakness. The clinical characteristics of this murmur are dealt with in detail at pp. 254–277, and its differential diagnosis at p. 215.

From what has just been said it will be seen that accentuation of the pulmonary second sound and increased loudness of the right ventricular first sound are merely signs of absolute overstrain of the right ventricle, and, while usually accompanying this type of dilatation, cannot be considered characteristic of it.

For a fuller discussion of the clinical features of the adolescent type of heart failure the reader is referred to the Essay on the Condition of the Heart in Anæmia, at p. 227.

How to Detect the First Signs of Cardiac Failure

Having now outlined the various symptoms and signs to which this type of cardiac dilatation gives rise, we are in a position to point out their practical application in answer to the question: 'How shall I detect the first signs of cardiac failure in a patient?'

Suppose it is a question whether an adolescent is overtaxing the heart by physical exertion of some sort, or whether a person who has had rheumatic fever is beginning to walk about before being really well enough to do so; or perhaps the question is whether a young woman suffering from anæmia had better take an active or a passive holiday. The answer depends upon the question: 'Is the right ventricle beginning to fail or not?'

The first sign of failure is breathlessness on exertion;

but this may not be present to a sufficient extent to be diagnostic. The pulse, too, may show little, if any, departure from the normal. We shall therefore have to turn to the examination of the heart.

1. The first unequivocal sign for which we look in such a case is an increase in the amount of the cardiac pulsation over the body of the right ventricle—i.e., over the fourth left interspace between the nipple line and the sternum. This shows commencing overaction of the right ventricle and will, if the case be one of absolute overstrain, be accompanied by some increase in the true loudness of the pulmonary second sound. A slight increase in the loudness of the first sound, as heard over the right ventricle, may also be noticeable if the case has been under careful observation previously.

In cases of relative overstrain—such as in a heart weakened by rheumatic fever—there may, or may not, be this increase in the loudness of the sounds. If the failure be primarily of the left ventricle and the right ventricular failure be secondary to this, an increased loudness of the sounds *will* be noticeable. If, on the other hand, the main cause of the increased pulsation be the undue distensibility of the right ventricle itself, there will be no increase in the loudness of the sounds it is producing.

In addition to the signs mentioned there will be some fullness of the neck veins—probably with some amount of false pulsation in them.

2. If the heart have reached a somewhat more advanced stage of failure, we shall find evidence that the weakest part of the wall of the right ventricle cannot withstand the intraventricular pressure and is beginning definitely to dilate.

In the distensible hearts of which we are speaking, this weakest part is the so-called conus arteriosus of the right ventricle; this—the weakest part of the ventricle—being dilated by the action of the stronger part.

These signs are increase of the cardiac dullness and the appearance of cardiac pulsation in the third left interspace.

3. The next series of phenomena are due to the further dilatation of the right ventricle upward.

They consist in the appearance of cardiac dullness, first relative and then absolute, in the second left interspace and, finally, of cardiac pulsation in this situation. Accompanying this dilatation there will be (where the overstrain of the right ventricle is absolute, not relative) the appearance of a pulmonary systolic murmur, together with increasing loudness of the pulmonary second sound and of the right ventricular first sound.

There will also be more marked fullness of the veins of the neck.

These phenomena may become extremely well marked in cases of anæmia, so much so that there may be dullness and pulsation for two inches or more in the second left interspace and the murmur may be so loud as to be conducted into the arteries of the neck (owing to the physical contact between the arch of the aorta and the pulmonary artery), and may also be accompanied by a vibratile thrill which is distinctly palpable in the second left interspace over the site of the pulmonary artery, and which is more marked when the patient is recumbent than when erect.

4. The next group of phenomena that would appear in a failing right ventricle are the phenomena which indicate that the main body of the right ventricle is giving way in the face of the extra blood pressure which it is called upon to develop. This group of phenomena does not occur, as a rule, in cases of relative overstrain, but only where the ventricle may be expected to be maintaining a blood pressure higher than normal.

This general dilatation of the right ventricle is associated with increase of the heart to the right—the well-known and usually described type of right ventricular dilatation. As has already been pointed out, this type of dilatation is more characteristic of the moderately distensible heart of adult life than of the distensible heart of adolescence, and its occurrence under the latter conditions would imply a very extreme degree of cardiac failure such as would occur in severe valvular disease.

The subject of dilatation of the ventricle to the right and the symptoms associated with it are dealt with later on when discussing cardiac failure in hearts of moderate distensiblity, and so no more need be said here on the subject.

EARLY DIAGNOSIS OF HEART FAILURE WITH ENLARGEMENT IN HEARTS OF MODERATE DISTENSIBILITY

We have now to take up the early diagnosis of heart failure in a moderately distensible heart, such as that usually found in early adult and middle life. The way in which the early signs differ from those seen in adolescence has already been outlined, and it has been pointed out that this difference appears to be due to the fact that the fibrous elements of the heart and pericardium, being newly formed and distensible in adolescence, are not able to exert much influence in checking over-distension of the organ; whereas in maturity and later life the restraining power of the fibrous tissues increases.

The main consequence of the increased strength of the fibrous elements is that the heart no longer has to depend mainly upon its muscular tissue for the prevention of over-distension and therefore the slight local variations in the muscular strength of its wall do not, in later life, determine the part which will be the first to yield to undue strain, as was the case in adolescence.

When the heart is depending mainly upon its fibrous tissues for the prevention of over-distension, the following points are noticeable.

In overstrain involving both sides of the heart—such as that from over-exertion, cycling, climbing, &c.—there is not a great disparity in the dilatability of the two sides, and we therefore expect to find dilatation of both the right and left ventricles.

Moreover, there is not the same differential yielding of the wall of the right ventricle that occurs in adolescence, but both the right and the left ventricle dilate in a very similar manner in the face of an excessive resistance to the outflow of blood during systole.

If the wall of the left ventricle be looked at, it will be seen that there is a decided difference between its thickness at the apex and round the base near the auriculo-ventricular orifice. If the relative thickness of the walls during ventricular systole be considered, it will be recognised that this difference will be accentuated. Now, suppose that the ventricle is working against an arterial resistance of increasing magnitude. A point can be reached where the resistance to be overcome requires a rise in the intraventricular pressure which is in excess of the resisting power of the thinner basal part of the wall, but is not too great for the expulsive power of the stronger apical part. In meeting such a resistance as this there will be a slight stretching of the weaker part of the wall with each recurring systole. The stronger apical part will in this way dilate the weaker basal part. The fact that this basal portion is the last to be emptied also involves a more prolonged strain upon it than upon the apical portions of the ventricle.

The over-distension of the basal portion involves another important change, and that is the over-stretching of the muscular and fibrous ring of the mitral valve. Stretching of the fibres of this ring will tend to the enlargement of the orifice and to mitral regurgitation. This, as is well known, is of the nature of a safety valve action, for the escape of blood into the auricle will lower the intraventricular pressure at the time of maximal strain and so lessen the tendency to further dilatation. Were it not for this, a dilatation of the ventricle once begun would, theoretically, continue unchecked until enormous *distension* resulted.

In the case of the right ventricle, the same sequence of phenomena will take place. The weaker basal portion being dilated by the stronger apical part and the dilatation being arrested by the production of incompetence of the tricuspid valve.

In adult life we often see well-marked dilatation of this basal portion of the right ventricle without any differential yielding of the slightly thinner part of the wall near the pulmonary valves.

Where this occurs, we are, I think, justified in saying that the fibrous tissues of the heart are taking the major part in preventing dilatation of the ventricle.

This type of dilatation may well be called THE ADULT TYPE of dilatation as distinct from the adolescent type already dealt with. As has already been pointed out, it is not intended to imply that in an adult this type of dilatation will always be found, because some degree of the adolescent distensibility of the heart may be retained in adult life. If these two types of dilatation are recognised, however, the distensibility of any particular heart can be judged by the degree in which it conforms to one or other of these types.

For instance, a heart showing slight dilatation of the conus arteriosus of the right ventricle and well-marked dilatation of the body of the ventricle must be taken as having nearly lost its adolescent distensibility. A heart with proportionally more dilatation of the thin part of the muscular wall must be a heart whose fibrous tissues are less rigid, and a heart showing less upward dilatation, in spite of well-marked dilatation to the right, must be a heart whose fibrous tissues have more resisting power.

The relative amount, therefore, to which one or other or both of these two types of dilatation is present (in any particular case of heart failure) gives important information as to the condition of the heart so far as the strength or otherwise of its fibrous elements is concerned.

In dealing with the early diagnosis of heart failure in hearts of moderate distensibility what has just been spoken of as the adult type of dilatation only will be considered, and the physical signs will be described which occur when the fibrous tissues are sufficiently resistant to prevent the adolescent type of dilatation from occurring at all.

Before, however, taking up the subject in detail it will be well to discuss shortly the distensibility of the heart in its bearing upon heart failure with dilatation in adult life.

Distensibility of the Heart in Adult Life

Although the age of the patient has been mentioned as roughly determining the amount of distensibility possessed by the heart, it cannot be so used in any absolute sense, for the distensibility in a series of patients of any particular age may vary considerably. In some patients the rigidity of the fibrous tissues of the heart appears to be much more marked than in others. Patients of seventy or eighty may be met with in whom the heart appears to be still quite distensible. For instance, I have known an old lady of over eighty live for years with a heart so large that its pulsation could be felt over the whole chest from the right nipple line up to the left axilla (*vide*, p. 320). On the other hand, patients of under fifty may show evidences of well-marked absence of distensibility.

When dealing with heart failure in adult life it is important to estimate the degree of rigidity which is present, because the management of the patients—the medicinal treatment and the prognosis—depend upon the distensibility of the heart.

This additional factor of fibrous rigidity complicates the question of heart failure. In adolescence the problem was a simple one, because there were only two points to consider—namely, the strength of the muscular wall and the resistance to the circulation which the heart had to overcome; therefore the amount of dilatation occurring was easily to be understood and interpreted by the study of these two factors.

The problem we have now to deal with is not so easy. For instance, suppose two hearts of equal muscular strength and working against the same circulatory resistance, but in the one case the fibrous tissues are more resistant than in the other.

The one with the less resistant fibrous tissues will dilate more than the other one will; yet it does not follow that the heart which does not dilate is the better organ of the two. Therefore, in dealing with a heart in adult and later life, we have before us, as it were, an equation with three quantities, two of which are unknown, and which we therefore cannot solve; instead of the equation with two quantities, one of which is known, which is presented to us by the adolescent heart.

As is the case with algebra, we must try to find a second equation whereby two out of our three unknown quantities may become known, and we fortunately can often succeed in estimating approximately two out of the three factors concerned —namely:—

1. Muscular strength of heart wall.
2. Degree of fibrous rigidity of the heart wall (that is, its distensibility).
3. The resistance against which the heart is working.

Therefore, when considering the early signs of cardiac failure in middle and later life, we cannot rely upon the amount of dilatation as a trustworthy guide to the degree of failure present as was the case in early life. We have to be on the look out for other signs of cardiac failure before we can appraise at its true value the presence or absence of enlargement of the heart in any particular case. This subject will, however, be further discussed when speaking of heart failure without enlargement, and the points raised will be better dealt with there than here.

At present, we shall simply deal with phenomena observable where a heart of moderate distensibility fails, and leave the question how best to recognise the degree of distensibility possessed by the heart until the question of heart failure without enlargement is discussed.

EARLY SIGNS OF FAILURE WITH ENLARGEMENT IN HEARTS OF MODERATE DISTENSIBILITY

CHANGES IN THE PULSE.—The first evidences of heart failure here, as in more distensible hearts, are to be found in the pulse, if the patient have been under observation before the heart failure set in and the normal character of the pulse be known. Under other circumstances, the pulse is a less certain guide in middle and later life than it was in early life, because of its character depending in part upon the condition of the arteries and not wholly upon the strength of the heart. One instance will suffice to illustrate the uncertainty which changes in the arterial wall or in the blood pressure bring about.

Take the case of a patient between fifty and sixty suffering from a severe attack of, say, abdominal pain, in whose case the question arises: Is there any temporary weakening of the heart? On examining the pulse it was found that the arterial walls were fairly soft and approximately normal, as judged by the finger, and that the pulse was regular and of fair volume and strength and of moderate tension.

Judging, therefore, by the condition of the pulse there was no marked weakening of the heart. When, however, the pain had passed off in a few hours' time, it was found that the man's usual pulse was very full and of high tension, and that during the attack of pain there had been a very considerable amount of temporary heart weakness. Had this man been first seen during a severe attack of influenza, or other condition accompanied by toxæmic affection of the heart muscle, an estimate of the presence or absence of heart weakness, based mainly on the pulse, might have led to a disastrous error in diagnosis.

Therefore the pulse must be used with caution as a guide in diagnosing the *absence* of heart failure in middle life. The further discussion of the indications given by the pulse will be postponed till the question of heart failure without enlargement is taken up (*vide* p. 59).

DYSPNŒA.—In this, as in other types of cardiac failure, breathlessness on exertion is one of the earliest and most important symptoms, but its characteristics are so well known that little need be said upon the subject here.

In the moderately distensible heart of middle life breathlessness is less often associated with sighing breathing than is the case in adolescence; and although the patient with dyspnœa can sometimes get relief from taking a long sighing breath, this type of breathing is not so well marked a phenomenon as it is in early life.

The diagnostic value of dyspnœa on exertion is somewhat lessened in middle life by the fact that it is more apt than in early life to be due to causes other than actual heart failure (*vide* p. 113).

On the other hand, in middle life, dyspnœa on exertion when certainly due to cardiac failure is of graver significance than it is in adolescence. This is due to the fact already pointed out that in the moderately distensible heart there is less disparity in the dilatability of the two sides of the heart than in the distensible one, and therefore, whereas in the distensible heart of adolescence dyspnœa can be caused by an amount of embarrassment which is too slight to interfere with the stronger left ventricle, in later life this is not the case, and dyspnœa is not likely to occur until the strain is great enough to interfere with the left ventricle as well as with the right.

PAIN.—Cardiac pain is a far commoner symptom of heart failure in middle and later life than it is in early life, but as it is especially characteristic of cases where cardiac failure occurs in relatively rigid hearts, its discussion will be postponed until this subject is dealt with (*vide* p. 82).

Another type of pain which gives important evidence of heart failure is that which occurs over the liver and is due to over-distension of the hepatic vein in tricuspid regurgitation; but it is a late rather than an early sign, except in the case of chronic heart failure, such as that due to valvular disease—where the sudden onset of pain over the liver may be an early sign that compensation is beginning to give way.

The general symptoms of dropsy and cyanosis occur as late rather than early signs of cardiac failure, and there is nothing of special importance to be noted about them in connection with the present subject.

PHYSICAL SIGNS of cardiac failure in hearts of moderate distensibility.—As has been already pointed out, the distinctive change in moderately distensible hearts, so far as the right ventricle is concerned, is a general yielding and distension of the wall around the tricuspid valve, causing a broadening of the heart towards the right, and also leading to systolic regurgitation of blood through the tricuspid valve.

The main clinical features of which we have now to speak result from these two phenomena.

Firstly, as regards the TRICUSPID REGURGITATION. The way in which tricuspid incompetence arises and its relationship to dilatation of the right ventricle have already been referred to. The phenomena due to the regurgitation of blood during the ventricular systole must now be studied. The simple statement that the regurgitating blood enters the right auricle and causes its ultimate dilatation and then causes a systolic distension of the veins—while summarising what often happens—does not express sufficiently accurately what, in all probability, takes place. The old view used to be that, after its contraction, the auricle was in a state of relaxation, and was therefore the unresisting recipient of any blood that was inclined to enter it. If this were the case the statement just made would be ample and accurate.

There is, however, good reason to believe that the auricular wall possesses a certain amount of tonic activity, even during its diastole, and there is little doubt that the walls of the veins, too, possess a large measure of tonic contractile power.

If this be so, the question as to the course taken by the blood when it regurgitates through the tricuspid valve may not be such a simple one, and may be stated as follows :—

If the tone of the auricular wall be good it may be possible for it to resist the sudden distension of the auricle by the blood which regurgitates through the tricuspid valve and so to deflect the stream of blood into the veins, causing their dilatation.

If, on the other hand, the muscular tone of the veins is better than that of the auricle the latter would be distended rather than the former.

But in speaking of the veins entering the auricle, we are speaking of the inferior as well as the superior cava, and the above statement must in consequence be amplified thus :—

When, owing to the better muscular tone of the right auricle or to the excessive amount of the tricuspid incompetence, the regurgitating blood tends to dilate the veins, it may pass either upwards into the superior cava or downwards into the inferior cava, or in both directions simultaneously. Now, clinically, it would appear that the Eustachian valve (with or without the assistance of the valves of the inferior vena cava and the muscularity of the vein) is able to offer more resistance to the downward passage of the regurgitating blood than can be offered by the tonic contraction of the superior cava (aided by the subclavian and other valves) to its upward passage, and that therefore it is more frequent for tricuspid regurgitation in its earlier stages to cause distension of the superior cava and its branches than of the inferior cava and liver.

Nevertheless, it is not very unusual to find that venous engorgement of the liver is present in the absence of any marked reflux into the superior cava, and therefore absence of tricuspid regurgitation cannot be diagnosed until the state of the liver has been carefully noted.

False Venous Pulsation.—With regard to the clinical phenomena of venous plethora due to tricuspid regurgitation, what has already been said as to false pulsation in the veins holds good also for the earlier stages of venous plethora in hearts of only moderate distensibility where tricuspid regurgitation is commencing.

In some cases of sudden failure of the right ventricle, as from a severe asthmatic seizure, extreme venous engorgement may be seen with well-marked false pulsation, but with no sign of true systolic pulsation.

As this subject has been dealt with at p. 14, and will be again referred to at p. 137, more need not be said here, except to point out that the feebler the muscular tone of the over-distended veins the more marked will be the false pulsation which is observable.

TRUE SYSTOLIC PULSATION IN THE VEINS.—When the pulsatile movements in the over-distended veins of the neck are carefully watched and timed by listening to the heart sounds or palpating the cardiac impulse, it is quite easy to distinguish true from false pulsation and to note the characteristic systolic filling of the veins from below upward.

In the slighter cases before the giving way of the subclavian and innominate valves it is necessary to note carefully the movements of the so-called jugular bulb at the root of the jugular vein, and of the innominate vein so far as it is palpable or visible in the episternal notch.

When the tricuspid regurgitation is more copious a well-marked systolic filling of the jugular and other veins of the neck will be noticeable.

When the tricuspid regurgitation is very extreme, and the muscular tone of the veins is good, their tenseness and the vigour of their pulsation may lead to their being mistaken for pulsating arteries and give rise to an error in diagnosis on the part of a superficial observer (*vide* pp. 87 and 222).

ENLARGEMENT OF THE LIVER.—Another well-known and important sign of failure of the right ventricle is enlargement of the liver, due to tricuspid regurgitation downwards into the hepatic vein. This is, however, a late rather than an early sign of cardiac failure, and need not be further referred to in connection with the present subject.

Physical Signs of Dilatation of the Right Ventricle.—As was the case in distensible hearts, the first sign that the right ventricle is having an abnormal amount of work to do is to be found in an increase of the cardiac impulse in the fourth left interspace over the body of the ventricle. An increase of cardiac pulsation here often gives the first sign of cardiac failure.

The next sign to appear is usually an increase in the cardiac dullness to the right of the sternum—at first without any discernable pulsation; but in cases showing well-marked dilatation some pulsation to the right of the sternum will be noticeable.

Before the dilatation has advanced as far as this there will probably be evidence that the ventricle is dilating downwards as well as to the right, as shown by the appearance of pulsation in the epigastrium. In some cases, indeed, the first sign may be the appearance of palpable or visible pulsation in this situation under the lower end of the sternum. When not very definite this may be best detected by laying the hand flat upon the lower part of the sternum and epigastrium—provided of course that the palm of the hand has been educated for purposes of palpation.

When dilatation to the right is well marked, it is easily found by percussion, if looked for; but in its lesser degrees it may be necessary to percuss the chest with the lungs fully deflated by deep expiration before it is possible to decide whether the right side of the heart is dilated or not. If a certain amount of emphysema be present it will most likely not be possible to decide with certainty whether the ventricle is dilated to the right or not. Under these circumstances, however, this is not of much consequence clinically, because the downward displacement of the heart which usually accompanies emphysema, when at all well marked, will bring the lower part of the ventricle within reach of palpation in the epigastrium, and the amount of cardiac failure present can be estimated by the amount of epigastric pulsation taken in conjunction with the amount of emphysema.

When speaking of the dilatation of the heart to the right, which is noticeable in heart failure, nothing so far has been said as to dilatation of the right auricle. As the right auricle lies to the right of the ventricle it would naturally be supposed that in cases of tricuspid regurgitation part of the increased area of dullness to the right of the sternum would be due to the dilatation of the right auricle. As a matter of clinical observation, however, it is extremely rare for a dilated right auricle to come sufficiently near to the anterior chest wall for it to be recognisable by palpation—or even, I believe, by percussion.

During the last twenty-five years, I have examined with the cardiograph almost every case I have met with where it seemed at all possible that pulsation to the right of the sternum was due to the right auricle; but with only two or three exceptions the cardiograph has shown conclusively that the pulsation was due to the right ventricle and not to the auricle. In only one case was it possible to obtain true auricular tracings, and in another case, such tracings as could be got suggested that the faint pulsation observable was due to the dilatation of the thin membranous part of the auricle near the inferior cava rather than to a dilatation of the muscular part of the auricle itself (*vide* pp. 339, 404, and Plate XII).

This absence of auricular pulsation in right-sided dilatation of the heart shows that dilatation of the right ventricle, as a rule, precedes dilatation of the right auricle. The stretching of the anterior wall of the right ventricle causes the auriculo-ventricular groove to be carried so far to the right of its normal position that the whole of the anterior aspect of the dilated heart is occupied by the right ventricle, and the dilated auricle projects simply towards the right and does not come into relationship to the anterior chest wall.

Post-mortem examination, where the heart is flaccid and undistended, may seem to controvert this statement; for it may seem as if the right auricle would come into relationship to the anterior chest wall during life. If, however, the right ventricle be distended by injection or by the simple and easier method of inserting a deflated thin rubber bag (penny toy balloon), and inflating it with air or water by means of a rubber tube, it can easily be shown that the above statement is correct, in the very great majority of instances, and that as a rule the right auricle when dilated does not come into close enough relationship to the anterior chest wall for its pulsation to be observable clinically, but that the pulsating portion of the right heart is almost invariably the right ventricle.

Although a dilated right auricle does not, as a rule, give signs recognisable by palpation on percussion anteriorly it may reveal its presence to auscultation posteriorly; for an abnormal distinctness in the cardiac sounds or murmurs, as heard over the lower right interspaces near the spine posteriorly, is often noticeable in cases where the right auricle is in all probability dilated. Such an increased loudness is to be expected owing to the anatomical position of the right auricle.

AUSCULTATION in heart failure where the heart is of moderate distensibility.—The information given by auscultation in cases of right ventricular failure is very valuable, and in middle and later life it has to be relied upon more than percussion or palpation. In adolescence, we saw that this was not so, and that palpation and percussion were of more diagnostic value than auscultation.

In the early stages of failure, important information can be gathered from the relative loudness of the heart sounds as heard at the apex (over the left ventricle) and over the right ventricle between the sternum and the nipple line. The loudness of the pulmonary second sound is also a very valuable guide to the amount of work which the right heart is doing, and its loudness over its point of maximal intensity in the second or third interspaces and over the body of the right ventricle must be compared with the normal and with the loudness of the second sound as heard at the apex, which is, as a rule, entirely due to the aortic portion of the second sound. The loudness of the second sound, as heard in the aortic area, while of considerable clinical value, is not of so much service for the comparative observations here spoken of, because its loudness is more dependent upon the condition and amount of the overlying lung—factors whose exact value it is not always easy to estimate.

MURMURS DUE TO TRICUSPID REGURGITATION

Of the murmurs which are due to tricuspid regurgitation, the most generally recognised is the systolic murmur produced at the tricuspid orifice; but it must be remembered that, while this is a very valuable evidence of regurgitation, when present, its absence is of no value as negative evidence; for it is extremely common for well-marked tricuspid regurgitation to be unaccompanied by this systolic murmur.

THE TRICUSPID SYSTOLIC MURMUR.—As regards its characteristics: its tone is somewhat lower as a rule than that of the mitral systolic murmur, as might be expected from the lower blood pressure on the right side of the heart as compared with the left.

Its point of maximal loudness, when not very well marked, is usually over the fourth and fifth interspaces just to the left of the sternum. Since the regurgitant stream, producing the murmur, is flowing towards the right into the auricle, it might be expected that the murmur would be first and best heard to the right of the sternum. This is, however, not so clinically, and the reason is that already given when speaking of the auricle in relation to palpation and percussion—namely, that it does not, as a rule, come near the anterior chest wall.

The ordinary rule as to the conduction of murmurs does, however, hold good for tricuspid regurgitation, as is shown by the rare cases where the right auricle can be examined. In the only case I have seen, not only was the tricuspid murmur very loudly conducted into the auricle, but the impact of the regurgitating blood was so forcible upon its antero-lateral wall that a very well-marked systolic thrill could be felt in the fourth right interspace internal to the nipple line. This thrill had its point of maximal intensity just internal to the nipple where the regurgitant stream of blood might be expected to strike the wall of the auricle.

When the murmur is louder its area of audition is an elongated oval reaching from about the third rib above to the sixth rib or so below, and from the left lateral edge of the sternum nearly to the left nipple line. When louder still, the area broadens to the right, embracing the whole of the sternum and also becoming audible to the right of it (*vide* figs. 76 and 110). When heard as widely as this, the true systolic tricuspid becomes merged with the next murmur of which we have to speak, although the two may still be more or less distinguishable by a difference in tone.

The tricuspid murmur is, when well marked, often audible downwards and to the right over the line of the inferior cava and the hepatic vein, and this direction of conduction is sometimes of value for its identification when other murmurs are present. The fact that on listening downwards and to the right the murmur retains its distinctness as compared with the first sound of the heart may be taken as strong evidence that a tricuspid systolic murmur is present. If, on the other hand, on listening in this direction the distinctness of the murmur is lost more rapidly than the distinctness of the first sound of the heart, it is highly probable that the murmur has its point of origin farther away than the right ventricle, and is a loudly conducted mitral or other systolic murmur (*vide* p. 279).

When the tricuspid regurgitation is considerable enough to cause a well-marked systolic impulse in the veins, the murmur will be well conducted into the veins of the neck and will merge into the murmur to be next described.

THE SUPERIOR VENA CAVA MURMUR of tricuspid regurgitation.—In addition to the ordinarily described murmur of tricuspid regurgitation, there is another one which is not as yet generally recognised, although it is of very common occurrence and is of very considerable diagnostic value.

The murmur in question is produced in the superior vena cava at the point where it passes through the pericardium, and it can only occur when there is sufficient regurgitation to cause marked distension of the vein above and below this point.

When the vein is distended, the strengthening of its walls by the fibrous tissue of the pericardium, at the spot where it passes through that structure, will limit the amount of dilatation in this situation, thus causing somewhat of a constriction in the dilated vein. The distension of a dilated superior vena cava, with hard paraffin, prior to the removal of the heart from the body, readily demonstrates this narrowing of the dilated vein, and shows it to be sufficient in extent to cause a murmur as the regurgitating blood rushes through into the more dilated vessel beyond.

Before noticing this peculiarity of a dilated superior cava, some twenty years ago, I had already come to recognise the presence of a systolic murmur audible in cases of tricuspid regurgitation whose point of maximal loudness was much to the right of that of the true tricuspid systolic murmur, and the discovery of the above-named peculiarity of a dilated superior vena cava showed how such a murmur might be produced.

Increased familiarity with this systolic murmur of venous origin has only confirmed my early observations and convinced me that there is justification for calling it the superior vena cava murmur.

As regards tone, it closely resembles that of the true tricuspid systolic, and when both murmurs are present, the only means of deciding as to its presence or absence is by noting whether there is or is not a second point of maximal intensity over the situation of the portion of the superior cava where the murmur is produced.

It is by this point of maximal loudness or intensity (its 'P.M.I.') that this murmur is best recognised, and by this it is distinguished from the tricuspid systolic murmur. It is heard best to the right of the sternum over the higher interspaces—usually the third and fourth, or the second and third—and usually it rapidly lessens in loudness on listening more than an inch from the sternum.

Sometimes the maximal loudness is higher—namely, in the second and first interspaces—and when this is the case the murmur will very easily be mistaken for one of aortic origin, owing not only to its P.M.I., but also to the fact of its conduction along the vessels. When a thrill is present in the veins, as occurs in some cases of extreme tricuspid regurgitation—the similarity between the two murmurs is very close—moreover, the strongly pulsating veins may simulate the pulsating arteries of aortic regurgitation (*vide* Essay IV, p. 220 and 222).

This murmur, when accompanied by a true systolic tricuspid murmur, is easy of recognition, for it is not at all well conducted downwards, and is often not heard at all over the cardiac area below the level of the fourth rib, neither is it conducted towards the liver. (For the further discussion of this murmur, *vide* p. 218.) The recognition of this murmur often gives very useful clinical information. For example, in the absence of recognisable dilatation of the heart and of a true tricuspid systolic murmur, its occurrence shows that there is really considerable dilatation of the superior cava. Moreover, its absence gives more certain evidence as to the absence of tricuspid regurgitation than does the absence of the true tricuspid systolic murmur.

The other auscultatory phenomena observable in cases of this type of dilatation of the right ventricle are of less clinical value; and as they will be fully discussed subsequently, they need only be mentioned here. They are a third sound produced in the right ventricle, and sometimes a right ventricular mid-diastolic murmur. These are most frequently noticed in cases with anæmia, and will be dealt with when heart failure in anæmia is discussed (*vide* p. 282).

When the diagnostic difficulties associated with dilatation of the right ventricle are taken up, the occurrence of a thrill in the veins due to tricuspid regurgitation and also the occurrence of a sound due to closure of the venous valves or to murmurs produced by their incompetence will be referred to (*vide* pp. 219 and 220).

CHANGES IN THE LEFT VENTRICLE IN HEART FAILURE WITH ENLARGEMENT

When speaking of the distensible heart of adolescence changes in the left ventricle were not touched upon because the relative weakness of the right heart in these cases prevented any early changes from taking place in the left. In anæmia this is especially the case, and even in simple overstrain a very considerable degree of heart failure with breathlessness can exist without any signs pointing definitely to left-side failure.

In the less distensible heart of middle life, however, this same disparity does not exist, and we expect to find some evidence of left-ventricle failure in every well-marked case of cardiac failure through overstrain, and sometimes the two sides of the heart appear to be equally affected quite early on.

In this connection it must not be forgotten that the most distensible basal portion of the left ventricle lies deeply in the thorax, and therefore changes due to dilatation may not show until they are well marked.

Firstly, as regards the pulmonary veins. The only means we have of judging as to their condition is by noting the amount of work which the right ventricle is doing; and where we find distinct evidence that the right heart is doing an abnormal amount of work, as judged by the loudness of its sounds and an increase in the amount of its pulsation, we are justified in inferring early failure of the left ventricle—provided that it is possible to exclude with reasonable certainty any hindrance to the circulation of pulmonary origin, such as emphysema, anæmia, &c.

As regards the left auricle, it, too, is of little value to us clinically as a guide to the condition of the ventricle from the point of view of the early diagnosis of heart failure, because of its being on the posterior aspect of the heart; nevertheless, it may, when much dilated, give some evidence both to percussion and auscultation, for under these circumstances it may show some recognisable dullness in the left interscapular region —as described by Dr. Ewart; and on listening in this region it is often possible to notice that the heart sounds are clearly audible, whereas normally they are usually not heard.

The abnormal conduction of the heart sounds to the back in this situation may be taken as pointing to dilatation of the auricle, and this is often of value as a confirmatory sign of dilatation, for it is noticeable with an amount of dilatation insufficient to cause any percussion dullness.

As to the left ventricle itself, the two chief signs of failure with enlargement are displacement outwards and downwards of the apex and the presence of a mitral systolic murmur. As regards the displacement of the apex, it is sometimes said that hypertrophy with dilatation causes an enlargement of the heart which carries the apex downwards and outwards in equal proportions, and therefore by causing a uniform symmetrical enlargement maintains the apex in the same axis as before; in dilatation without hypertrophy, on the other hand, there is more broadening of the ventricle, and therefore the apex is carried rather more outwards than downwards, and so takes up a position to the left of the normal axis. Although this may be so, to some extent, the broadening is more apt to be marked above the apex than to involve the apex itself. As regards the signs of heart failure given by the loudness of the left ventricular sounds, no generalisation can be made, because if the failure be due to muscular weakening from nervous or nutritional causes, the sounds will become weaker and shorter and wanting their full tone. If on the other hand it is because of absolute overstrain from rise in arterial resistance or excessive exertion, the heart failure may be accompanied by undue loudness. But if, as is possible, these two factors be nicely balanced, heart failure may be accompanied by absolutely normal loudness of the heart sounds.

It is not until the ventricle is sufficiently dilated to produce some incompetence of the mitral valve that we have the first unequivocal sign of left ventricular dilatation—namely, the mitral systolic murmur.

It is sometimes not easy to decide whether a faint murmur at the apex be really due to mitral regurgitation or not; but this point can usually be definitely cleared up by noting the way in which the murmur is conducted towards the axilla as compared with the conduction of the first sound. If the murmur arise at the mitral valve it will retain its relative loudness as compared with the first sound.

If the murmur be fainter than the first sound, as heard at the apex, when listening farther and farther out, a point may be reached—say, in the mid- or the posterior-axillary line—when the first sound only is audible and not the murmur; but, nevertheless, the gradual and equal diminution in the loudness of the two sounds, until the one is too faint to be heard, shows that they both arise at the same valve and that the murmur is really a mitral systolic.

Again, supposing they are equally loud: the fact that the murmur is audible wherever the left ventricle first sound is audible will prove its mitral origin.

But, on the other hand, supposing that the murmur is fairly loud at the apex, but that on listening towards the axilla it rapidly becomes fainter and is lost, while the first sound remains fairly loud, the diagnosis can certainly be made that this is not a mitral murmur showing incompetence of the valve.

The diagnosis of one of the most puzzling forms of cardio-respiratory murmur depends upon this method. Sometimes a murmur exactly resembling a mitral systolic murmur is caused by pressure of the apex of the heart upon the adjacent lung, and it may even continue to be produced when the breath is held. Careful study, however, of its loudness in relation to that of the first sound will show that its point of maximal loudness is some inch or so external to that of the first sound. On studying the conduction of this murmur, outwards from the apex (where the first sound is loudest) towards the axilla, it will be found that it increases in loudness for the first inch or so—i.e., until a certain amount of lung intervenes between the stethoscope and the heart—showing that the murmur arises in this portion of the lung and not in the heart itself. In such a case, although the murmur may be audible round to the back and the angle of the scapular, its pulmonary origin can be proved by watching the way the point of maximal intensity shifts during full inspiration and full expiration—always keeping an inch or so away from the uncovered portion of the heart and never heard best over the heart itself (*vide* p. 446).

As regards the sequence of the phenomena showing heart failure in moderately distensible hearts, it is not possible to make any useful generalisation as was the case in adolescence, because the mode of onset is so liable to vary, according to the degree of distensibility which the heart possesses. The chief guides are the well-known ones—namely, the amount and nature of the venous pulsation in the neck, the amount of dilatation of the heart to the right and to the left, and the presence or absence of a murmur due to tricuspid regurgitation, produced either at the valve itself or in the superior cava, and the presence or absence of a mitral systolic murmur. The presence of epigastric pulsation, and enlargement and pulsation of the liver, must also be looked for and its degree noted.

HEART FAILURE WITHOUT ENLARGEMENT

We now come to the second main group into which it is necessary to divide all cases of heart failure. In the group with enlargement just discussed it was pointed out that dilatation could only occur provided that the internal pressure which the heart muscle could develop was in excess of the resisting power of the weakest portion of the ventricle wall.

As soon as these conditions are not fulfilled, owing to abnormal weakness of the heart muscle or abnormal resisting power on the part of the heart walls, failure with enlargement is no longer possible, and heart failure without enlargement will occur instead.

BOSTON MEDICAL LIBRARY NOV 15 1915

It may perhaps be objected that where the heart muscle reaches this degree of relative weakness the circulation cannot be carried on and death must occur; but this objection is answered by clinical observations which show that there is a fairly wide margin between these two alternatives, and that life—and sometimes a fairly active life—*is* possible under these circumstances.

This group must be divided up into two sub-groups according to the distensibility possessed by the heart; for here, even more than in the last group, the signs and symptoms of cardiac failure depend upon the degree of elasticity of the heart, and the phenomena observable in the muscularly weak adolescent heart are quite different from those seen in the relatively rigid heart of old age.

In this case there will be no need, as in the last instance, to discuss the subject under three headings, according to the degree of distensibility which the heart possesses, because hearts of moderate distensibility do not show phenomena differing from those at the two extreme ends of the series, but only a combination, to a varying extent, of these phenomena.

In approaching this subject one of two courses might be taken: either the reader might be led step by step through the clinical observations which led the writer up to the theoretical considerations which will here be given (and it is of interest to note that some of the most obvious of these latter were the last instead of the first to be deduced from the clinical facts); the other course, and probably the best one, is to present the subject as complete as possible from a clinical and theoretical point of view, irrespective of the stages the writer passed through in the evolution of the theory here given.

To take the second of these two courses will probably give the most generally useful review of the changes in the heart with which it is now proposed to deal.

Take now the case of an elastic, distensible heart—such as is found in an adolescent of the age, say, of sixteen or seventeen. We have discussed what happened when the intraventricular pressure, owing to muscular weakness, falls so much below its normal level that it is no longer able to expel the normal contents against the pressure which it has to face in the arteries.

Let us take the case of the left ventricle in, say, a case of rheumatic myocardial weakness or in the toxic heart failure that occurs after influenza or diphtheria.

As soon as the muscular power falls so low that it cannot expel the normal ventricular charge (of about, say, 3 ounces of blood) against the resistance it has to face in the aorta, one of two things must happen: either the patient must die of asystole or nature must find some means of lessening the load proportionally to the muscular power of the heart.

Now, if the physical and physiological conditions we are dealing with be looked into, a way out of the difficulty is evident; for it is clear that a muscular chamber which cannot commence the act of emptying itself when it is full and its muscular fibres are fully stretched, might nevertheless be able to do so when it was not so fully distended and the muscular fibres of its walls were able to support one another more. The muscular walls of a partially filled ventricle are thicker, and therefore presumably more effective, than those of a fully distended one.

It is probable also that the principle of the hydraulic press applies here to some extent, and that, taking the diameter of the aorta as a fixed quantity, the ease with which the ventricle empties itself into the aorta will be proportional to the smallness of its sectional area as compared with that of the aorta.

Apart from these theroretical considerations the clinical fact is certain that in cases of extreme myocardial failure the heart does diminish in size, and the volume of the thoracic contents and of the liver also diminish, thus showing a diminution in the amount of blood which the heart is able to keep in active circulation.

The mechanism whereby this diminution is brought about is quite simple, for those who hold—as the writer has done for more than a quarter of a century—that the ventricle fills itself by an act of true muscular expansion, or, in other words, that the ordinary longitudinal contraction of the muscular fibres is followed, in the shortened and broadened fibres, by a transverse contraction whose effect is suddenly to restore the fibre to the condition it was in prior to the contraction.

In the adolescent, as has already been pointed out, the fibrous elements of the heart are soft and possess a maximal degree of distensibility and a minimal degree of rigidity; therefore in adolescence their power of resisting deformation must be at its minimum, and the heart will have but little tendency to return to its former shape, after contraction, in virtue of its elastic recoil alone.

Therefore, the heart's power of *mechanical* aspiration, after contraction, will not be great, and the heart will have to depend mainly upon its muscular power for the expansile force whereby it fills itself.

Now in the cases we are considering, the muscles have lost much of their contractile power through inflammatory or other damage, and this being so their muscular expansile power must also have suffered. In these facts, I think we can see the means whereby, in severe myocardial failure, the work of the heart is compensatorily cut down proportionally to the muscular weakening. The same heart weakness which prevents the heart from emptying itself, also prevents it from filling itself.

This statement ought, however, to be reversed, and read thus :—

The first result of extreme muscular weakness in a heart whose fibrous tissues are soft and possess little resisting power is that the heart is unable properly to fill itself, because its power of filling itself is dependent solely upon the aspiratory force which the active expansion of its muscular walls can develop. By this failure to fill itself properly the heart is largely safeguarded against the risk of asystole, for the amount of blood which the heart draws into itself is proportional to the amount which it is capable of expelling.

As the expansion movement is a weaker one than contraction, suction will fail sooner than expulsion, and therefore there is no likelihood of the heart drawing into itself more blood than it is able to expel.

To those who do not fully accept the muscular-expansion theory this diminution in the volume of the heart and of the blood in active circulation is also explicable; for the gradual onset of muscular weakness will limit the volume of the circulation. The weakness of the right ventricle will lessen the volume of the pulmonary circulation and, as a consequence, the amount of blood available for the filling of the left heart will also be lessened, and the deficient amount of blood forced into the systemic circulation will presumably lessen the venous return to the right ventricle, an abnormal amount of blood being left in the venous reservoirs.

In the distensible heart of adolescence this is what takes place when severe myocardial failure sets in, and this is the reason for the smallness of the heart and the diminution in the size of the vascular organs, lungs, and liver, which is clinically observable in such cases (*vide* pp. 68, 118, 180, 185).

In contrast with these cases, let us briefly review what takes place in a heart whose tissues have attained to the increased rigidity which comes with advancing years. In such a heart, we are, I think, justified in expecting that the non-muscular portion of the heart has more power of resisting deformation than is the case in the still growing heart of adolescence: in other words, in the adult the heart not only possesses the power of enlarging itself by its muscular recoil movement, but also tends to return to it original size after contraction by reason of a *mechanical* elastic recoil as well.

Therefore, in the adult, weakening of the heart muscle interferes with its expulsive power more than it does with its aspiratory power, and a weak heart is able, by virtue of its mechanical elastic recoil, to draw into itself a larger charge than its muscular power is able to expel. This is, in my opinion, the explanation of the well-known fact that sudden death from asystole is a far more common result of muscular weakness in later life than it is in early life.

Before going further into this latter question, however, the simpler subject of heart failure in distensible hearts will be discussed.

HEART FAILURE WITH SMALL HEART

In the type of heart we are now dealing with, well-marked muscular failure of the heart is usually associated with an actual diminution in its size, so far as it is clinically observable, and therefore the term 'heart failure with small heart' is applicable to cases in this group. This fact was first brought to the writer's notice by the careful study of the size of the heart and liver in cases of malnutrition, as is recounted in Essay II, and by finding that the starvation necessary for the treatment of gastric ulcer did cause a shrinkage of the more elastic of the vascular organs, betokening a diminution in the amount of blood filling them, and therefore in the total volume of the blood. From this, he was led on to the observation that a similar diminution in the size of the liver and the lungs and also of the heart was noticeable in cases where the heart was muscularly too feeble to keep a normal amount of blood in circulation. The theoretical considerations here given were only slowly arrived at after the certainty of the diminution in the size of the heart and in the volume of the circulation was evident as a clinical fact.

When, therefore, we are speaking of distensible hearts, it can be asserted that 'whenever, owing to muscular weakness, the expulsive power falls below the resisting power of the weakest portion of the ventricle walls heart failure occurs without dilatation; and, further, whenever the expulsive power of the ventricle falls below the resistance to be overcome in the aorta, diminution in the amount of the filling of the heart will take place, together with a lessening of the total amount of blood in active circulation—a condition well described as 'heart failure with small heart.'

This group of cases of heart failure with small heart is not a large one, but embraces a by no means unimportant type of case, where there is extreme muscular weakness, due to such causes as myocarditis, toxæmic poisoning from such diseases as rheumatism, diphtheria, influenza, &c. To some clinicians the occurrence of such a group at all may be a matter of doubt; but if they will only follow the arguments and the clinical observations detailed in this and the succeeding essays and examine carefully, for themselves, cases similar to those recorded here, the existence of this group will become for them a matter of the same certainty as it is in the case of the writer.

The careful study of the state of the circulation and of the size of the heart, and of the more elastic and vascular of the viscera, in cases of extreme heart weakness, shows quite conclusively the presence of phenomena which indicate clearly that the total volume of the blood circulated by the heart has been compensatorily lessened, thus making it better proportioned to the lessened cardiac strength.

When the heart becomes too weak, owing to extreme muscular failure, to keep the normal amount of blood in active circulation a certain amount is cut out by the compensatory mechanism already described, and only an amount such as the heart can cope with is retained in the main arteries and veins.

What happens to the remainder of the blood cannot be definitely stated, but no doubt it is stored up in the usual reservoirs where reserve blood is kept (namely, the abdominal and other plexuses of veins) until such time as the heart recovers itself. Or, in the case of persisting muscular weakness, until a compensatory diminution in the total blood volume can be established. The theoretical consideration of this subject is dealt with in succeeding essays and also the clinical evidence that there is a diminution in the size of organs—such as the lungs and liver—in cases of extreme muscular weakness of the heart; therefore, the clinical features only of extreme heart failure with small heart will be here discussed.

Before, however, taking up the early signs of this type of heart failure it will be well to discuss its clinical characteristics, where fully established, in order that a fairly complete clinical picture of the heart and circulation may be given.

A careful examination of the heart and circulation in a well-marked case of extreme muscular asthenia, from such a cause as myocarditis or toxæmia accompanying rheumatic fever, or following such a condition as influenza or diphtheria, will bring out some or all of the following phenomena. Firstly, as regards general and well-known signs. The pulse will become weak and small. The heart sounds will lessen in loudness and vigour, and the apex beat will become faint or indistinguishable.

Amongst the more important of the less-known symptoms the following are of importance:—

1. The area of cardiac dullness will diminish in size as the strength of the heart fails. In even a moderately severe case of this type the absolute cardiac dullness may be almost or entirely absent and the relative dullness markedly lessened in size.

In severe cases of cardiac weakness it is by no means unusual to find that even the relative cardiac dullness is almost or entirely absent when the patient is lying back in bed, and these cases can assume no other attitude owing to extreme weakness and the tendency to fainting.

In such a case, the full lung resonance will come down to the full gastric resonance, in and internal to the left nipple line (for in such a case as this, the dullness of the left lobe of the liver will also be entirely absent (*vide* p. 68, and figs. 16–41).

The first comment that will be made upon this statement will be that for some cause or other the lungs are unduly large and cover up the heart. Examination of the size of the lungs will disprove this alternative, for percussion of the lower limits of the lung resonance show that the diaphragm is above the normal level, and its rise shows that there must be a distinct lessening of the total volume of the lungs (*vide* p. 68), and therefore the disappearance of the cardiac dullness must be due to a diminution in the size of the heart or a falling backward of the organ into the chest. Probably both causes are at work, for the underfilled organ might be expected to fall somewhat back into the chest under the action of gravity when the patient is lying back in bed.

I have not taken the circumference of the chest in these cases nor measured the angles formed by the ribs in order to obtain, if possible, further confirmation of the diminished content of the thorax; but it is quite probable that such observations would only give a negative result, the configuration of the chest wall being normal because of the compensatory change being one involving the diaphragm only.

2. The clinical evidences of a rise in the average level of the diaphragm constitute another series of signs of cardiac failure of this type.

A rise in the level of the gastric resonance is one of the most important and most often observed of these. In a shrinkage of the thoracic contents, including the heart, the left half of the diaphragm would naturally rise sooner than the right half, because the inertia of the liver would tend to retain that side in its normal situation. The gaseous contents of the stomach on the other hand would have no restraining influence upon the rise of the left half of the diaphragm and might possibly have the reverse effect. As this question of the rise of the diaphragm is fully discussed in Essay II, at p. 163, nothing more need be said here except perhaps that the rise can be proved to be due to a lessening in the volume of the intrathoracic contents, and that it must be due to a lessened fullness of the heart and blood-vessels, especially those of the lungs.

A rise in the upper level of the liver dullness on the right side is sometimes of value as indicating a rise of the diaphragm, in spite of the fact that it is less marked as a rule than the rise in the gastric resonance. It is sometimes extreme in degree (*vide* p. 82, and fig. 49).

3. Emptiness of the veins.

In cases of acute heart failure, where the heart muscle is not extremely weak, we are accustomed to look for the fullness of the veins of the neck, which results from tricuspid incompetence, and when in such cases it is absent we look confidently for the dilatation of the liver, which results from undue fullness of the inferior vena cava.

Where, however, the heart muscle *is* extremely weak, the reverse is the case; for the veins of the neck are empty, and the supraclavicular fossæ deeper than normal, and there is no sign of any distension of the liver, but rather the reverse.

4. Another sign of heart failure which is of value in measuring the muscular strength of the heart is the size of the liver dullness as judged by percussion when the patient is recumbent.

In muscular failure of the heart there is diminution of the area of liver dullness, and in cases of extreme heart failure this may be so marked that there is almost no absolute liver dullness anteriorly when the patient is lying back, and it is not at all infrequent to find that there is no absolute liver dullness anterior to the right nipple line, and that in front of this line the gastric or intestinal resonance meets the resonance due to the lung with no intervening absolutely dull area such as is found in the normal chest.

This absence of dullness is due in part to diminution in the size of liver, from its vessels being underfilled with blood, and in part to a want of rigidity due to the same cause which allows it to fall away from the chest wall, and in part to altered conductivity to percussion vibrations. This condition of the liver is also found when the blood volume is diminished from causes other than heart failure, and is fully discussed at p. 202.

Some of these phenomena must now be discussed more in detail.

CHANGES IN THE PULSE in heart weakness.—Changes in the pulse may be indicative of the onset of extreme muscular failure of the heart.

One of the most important changes indicative of weakening of the heart muscle is the often described sudden drop in the strength and volume of the pulse wave. This weakening of the pulse—in an attack of rheumatism, for instance—suggests inflammatory damage of the heart by peri- or myo-carditis. So far as the rate is concerned a sudden slowing of the pulse is very suggestive of toxic poisoning of the heart muscle in cases where the patient is suffering from some infective complaint such as influenza. Increase in the rapidity is not so sure an indication.

A more important sign and one which points strongly to myocardial weakness is a rise in the tension of the arterial wall (as measured by the fullness of the pulse between the beats), associated with marked feebleness of the pulse and lowness of the blood pressure—i.e., a weak pulse of relatively high tension. In such a pulse as this the artery remains quite full between the beats and, if gentle enough pressure be used, can be rolled under the finger as a continuous soft cord and does not subside as a normal pulse does.

The contracted state of the arterioles which this type of pulse implies is, in all probability a compensatory phenomenon. The heart is too feeble to keep much blood in circulation, or to maintain it at anywhere near the normal pressure, and therefore the systemic arterioles are kept in the greatest degree of contraction that the weakened heart can stand.

This serves the double purpose of diverting a maximal amount of blood from the systemic circulation to the brain and heart and also of maintaining the blood pressure at the highest possible level throughout the whole of the diastole, thus enhancing as much as possible the effect of the feeble systolic rise in the blood pressure.

In enteric fever this pulse of low blood pressure with relatively high arterial tension can very frequently be noticed, and it speaks of the degree to which the heart muscle has been weakened by the action of the specific toxin.

Such a pulse as this will suggest caution in the use of cardiac stimulants. Flogging an exhausted horse may result in his falling dead on the road.

Changes in Heart Sounds, &c.—Diminution in the loudness of the heart sounds is a valuable indication of weakening of the heart's action, and is always present in muscular failure. The marked weakening of the first sound of the heart which is so characteristic of severe enteric fever is a well-known indication that the heart muscle is being damaged by the enteric toxin.

Sometimes when one ventricle is more involved than the other in a case of fatty degeneration, the relative weakness of the sounds on that side of the heart is a useful guide.

I have more than once known marked weakness of the right ventricular sounds confirm the diagnosis of fatty degeneration mainly limited to the right ventricle, in cases of marked breathlessness combined with a good pulse and good left ventricular heart sounds.

Diminution of the area of cardiac dullness, especially if accompanied by weakening of the cardiac impulse, is an important sign of weakness of the heart muscle.

The diminution takes place on all sides of the cardiac area. The heart presumably shrinks in volume, and so the lungs encroach upon it. Instead of its normal shape the cardiac dullness assumes a semi-ovoid shape, and its lower border rises above the normal level: for the lower edge of the cardiac dullness is encroached upon by the rise in the gastric resonance which is found in such cases.

The cardiac dullness does not as a rule reach to the sternum unless it be for half an inch or so at its lowest part. This change in the area of the cardiac dullness is well shown in figs. 41-51.

Reference to p. 202 will, however, show that diminution in the area of the cardiac dullness accompanied by a rise in the diaphragm may be due to causes other than muscular failure of the heart, and therefore this sign cannot be taken by itself as a sign of heart failure. The sudden onset, however, of such a diminution, in a case where sudden loss of blood or malnutrition could be excluded, would suggest the occurrence of muscular weakness of the heart.

Other signs of CARDIAC WEAKNESS.—The remaining signs of weak heart muscle—namely, the rise in the gastric resonance and rise in the upper level of the liver which result from a rise in the level of the diaphragm, and also the diminution of the area of the liver dullness, are dealt with in Essays II and III. They are signs of lessened blood supply, and are also met with in cases of hæmorrhage or shrinkage in the volume of the circulating blood from other causes, and are therefore not pathognomonic of myocardial weakness.

Emptiness of the veins of the neck is an easily observed sign, and its presence is often of considerable value in deciding whether there is myocardial weakness or not. It must never be forgotten, however, that in ordinary heart failure in valvular disease the veins of the neck may be relatively empty, in spite of the presence of tricuspid regurgitation, where the Eustachian valve has given way and the blood which regurgitates from the right ventricle passes down the inferior vena cava into the liver, instead of into the neck veins. A very cursory examination of the liver will suffice to show whether the emptiness of the neck veins be due to this cause or not.

RIGHT VENTRICLE in muscular weakness.—Before leaving the consideration of cardiac failure with small heart, the question may be asked: Does compensatory diminution in the size of the ventricle occur on the right side of the heart as a primary phenomenon?

As a secondary phenomenon it must occur, for the right side of the heart is fed by the left.

All the evidence points against the right ventricle ever diminishing in volume owing to a primary failure in its muscular power.

The right side of the heart is more dilatable than the left, and dilatation leads to escape of the blood out of the heart altogether into the veins and not simply into the auricle and the closed pulmonary veins, from whence it is certain to re-enter the ventricle during its next expansion phase, as is the case with the left side of the heart.

A primary compensatory diminution in the size of the right ventricle is not therefore necessary, because of the ease with which blood escapes from an embarrassed right ventricle.

Under certain pathological conditions this safety-valve action of the tricuspid mechanism is largely in abeyance; but under these circumstances sudden death is apt to occur when there is over-distension of the ventricle, and I have not yet been able to detect any sign of compensatory diminution in the circulation similar to that which occurs in weakness of the left side of the heart.

The cases referred to are those where the distensibility of the right side of the heart is lessened by pericardial adhesions, and also more especially by adhesions of the visceral pleura to the sternum and ribs, anteriorly, as well as over the vertebræ posteriorly. This adhesion of the pleura anteriorly and posteriorly forms an inelastic fibrous wall to the right of the heart. This type of pleural adhesion is frequently the cause of sudden death under chloroform anæsthesia.

As a rule, however, the right ventricle is not faced with the risk of asystole, like the left ventricle, owing to the free leakage through the tricuspid valve, and the distensibility of the right auricle and great veins. In cases of fatty degeneration, limited more or less to the right side of the heart, the symptoms are not those dealt with in this essay.

The appearance of the above phenomena must therefore be looked for in a case where acute myocardial failure is to be anticipated, but more especially the signs concerning the pulse, the size of the heart and strength of its impulse, the size of the hepatic dullness, and the altered level of the gastric resonance.

DIMINUTION IN THE SIZE OF THE HEART AS A SIGN OF HEART FAILURE IN VALVULAR DISEASE, &c.

Before leaving the subject of heart failure with small heart it will be well to refer to the phenomena of a similar character which sometimes occur as a complication of valvular disease, and other cases of cardiac failure with which dilatation is associated.

In such cases, we must remember that acute or gradual failure in the vitality of the heart muscle may be associated with a diminution in the size of the heart and liver. Now, considering that dilatation of the heart and enlargement of the liver are the two most important signs of heart failure, the meaning of such a diminution may be mistaken, and the mistake may prove of very serious import to the patient. Thus in a critical case of heart failure with dilated heart, I have noticed a diminution to the extent of over an inch in the apparent size of the greatly enlarged liver taking place in forty-eight hours. This was no case of improvement, but a sign of heart failure due to over-stimulation; and a change in the medicinal treatment caused a return of the liver to its former size in another forty-eight hours, together with signs of improved cardiac action. A mistaken interpretation in this case leading to a continuation of the cardiac stimulant would probably have proved most disastrous for the patient (see p. 201).

A similar mistake may be made in the case of a heart which, owing to myocardial weakness, does not dilate when overstrained by such a condition as acute pneumonia. A normal or a small heart under such circumstances may be a sign of dangerous heart failure.

Dilatation of a Weak Heart a Sign of Returning Strength

We have just been drawing attention to the fact that diminution in the size of the heart and liver may in exceptional cases have an opposite interpretation to that which it usually bears.

In a similar way, dilatation of the heart may not infrequently have to bear an explanation the reverse of that which is usually applicable to it; for in the case of small heart, due to myocardial weakness (of which we have been speaking), one of the early signs of returning vigour may not only be a return to its normal size, but also an actual dilatation.

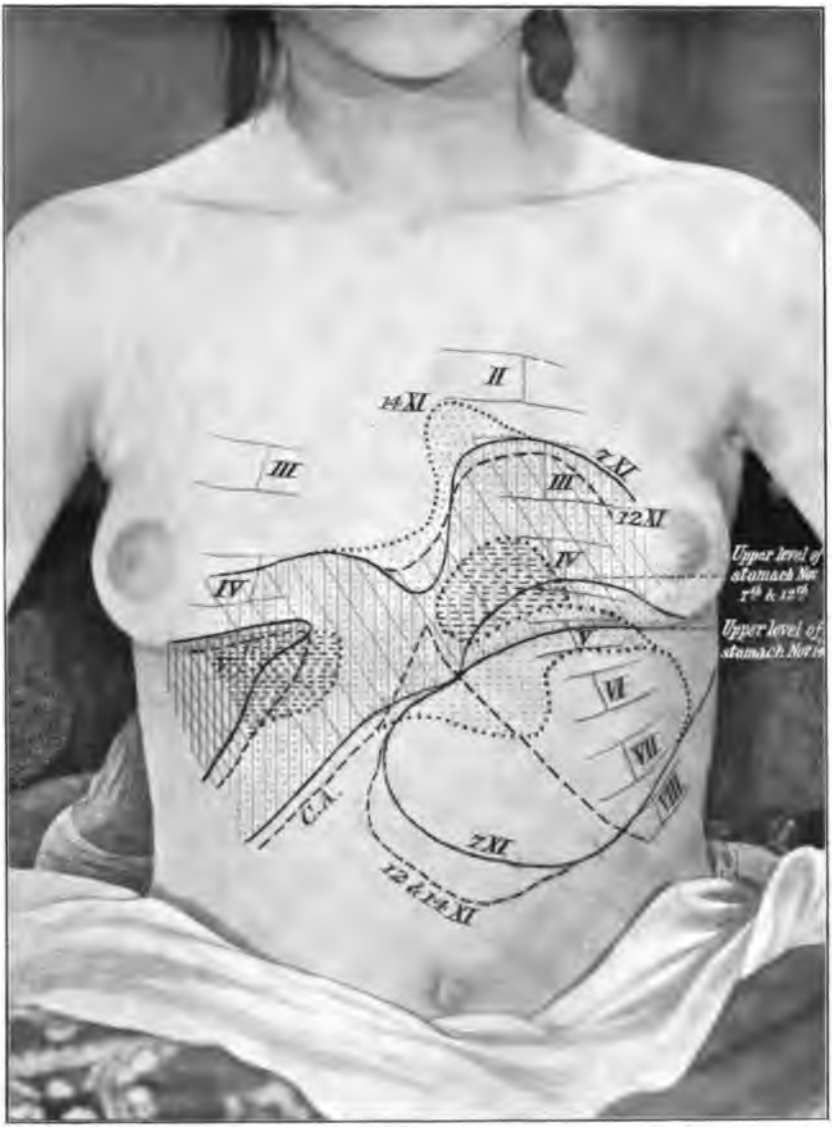
Such a dilatation must be looked upon as a satisfactory sign in such a case (*vide* p. 195, and figs. 42–52).

As an illustration of the way in which the progress of a weak heart may be observed by recourse to these methods of physical examination, I would refer to the series of diagrams given on pp. 66 and 100, and also to Plate I.

The following case is of great interest in this connection, for its shows very well how the principles of diagnosis here laid down are of service in clearing up a somewhat difficult case of heart failure.

The patient, a man of about forty years of age, had an attack of rheumatic fever eight months prior to my seeing him, and ever since had suffered from dyspnœa with occasionally some dropsy of his ankles. His breathlessness was considerable, for he could only walk up some five or six steps at a usual pace, and in order to do a whole flight, he would have to go very slowly. The man was rather pale, and it was stated that when he over-exerted himself, he was apt to go very pale. On examination it was found that the veins of the neck were quite empty when standing up, but filled up slightly when he lay down, showing some false pulsation, but no signs of tricuspid regurgitation. The liver dullness to the right was practically normal, and also showed no signs of regurgitation into the inferior vena cava. The pulsation in the jugular bulb was about normal in volume and of fair strength. The heart itself was not enlarged and was apparently smaller than normal, for, although the apex could be just felt in about the nipple line in the fifth interspace when he was standing up, when he lay down there was well-marked gastric resonance in the fifth space as far as the nipple line, and there was very little, if any, pulsation to be felt. There was no absolute cardiac dullness, and the relative dullness was only to be found in the fourth, and for about the normal extent in the third, interspace. The sternum was resonant, and the dullness of the left lobe of the liver was absent. As regards the heart sounds there was at the apex a well-marked mitral systolic murmur accompanying a short and not very loud first sound. The second sound was also rather short and sharp. On listening over the right ventricle it was found that there was no evidence of marked overaction as would have been expected considering his breathlessness and the presence of evident mitral regurgitation. The sounds were of only moderate loudness, and no pulsation could be felt. The pulse was of moderate volume and strength and was markedly irregular. The cardiac irregularity was of the type that we now associate with auricular fibrillation.

PLATE I


II
14 XI
III
7 XI
III
12 XI
Upper level of stomach Nov 7th & 12th
IV
IV
Upper level of stomach Nov 14
V
VI
VII
VIII
C.A.
7 XI
12 & 14 XI

A Photograph of the Chest, showing the rapid return of the Heart and Liver Dullness towards Normal after a diminution in size due to Cardiac Weakness.

The results of percussion on November 7, 12, and 14, 1911, are here shown. The ribs are marked in roman numerals, and the costal arch (C.A.) by a broken line. The outline of the organs on the 7th is shown by a continuous line, on the 12th by a broken line, and on the 14th by a dotted line. For the sake of clearness, shading has been added—consisting of vertical lines over the cardiac and liver areas as they were on the 7th, oblique lines for the 12th, and dotted shading for the 14th. Short dark lines are used for the absolute dullness on November 14, and only the anterior part of the absolute liver dullness is shaded. Note the reappearance of the absolute cardiac dullness and of the relative dullness of the left lobe of the liver on November 14, although the gastric resonance was still obtainable in the fourth interspace. Note also the upward dilatation of the heart into the second interspace on the 14th, although the patient was still confined to bed.

The diagnosis here was mitral incompetence, with considerable myocardial weakness and also auricular fibrillation (see fig. 1).

The diagnosis depended upon the fact that, in spite of the mitral regurgitation and breathlessness, there was no dilatation or hypertrophy, the heart being, on the contrary, smaller than normal, as was shown by the high left diaphragm and the lessened area of cardiac dullness; nor, in spite of the faulty action of the left ventricle, was there any compensatory overaction of the right ventricle. Another evidence of weak muscular action was the fact that, in spite of the cardiac failure and breathlessness, there was no over-distension of the veins, and the dyspnœa was accompanied by pallor rather than by cyanosis when he over-exerted himself.

In this case, then, the rheumatic fever appeared to have done more damage to the heart wall than to the valves (although there was some mitral incompetence), and the damage to the heart wall seemed also to have caused auricular fibrillation.

So far as the question of rigidity of the heart wall was concerned, it was only possible to say that there was no evidence of any abnormal rigidity, and that the diminution in the size of the heart pointed to there being none. The absence of dilatation was sufficiently explained by the amount of muscular weakness present.

The above estimate of the patient's condition, which was made at his first visit on August 21, 1913, was confirmed by the after-history of the case, which was, shortly, as follows:—

He was seen a second time, on September 11, and it was found that the heart was larger (as shown in fig. 2), and the diaphragm lower. The apex was now 4½ inches from the sternum in the fifth interspace, instead of being in the fourth interspace and under 3 inches from the sternum. The patient stated that he was stronger and less breathless on exertion. The heart, though still irregular enough to suggest auricular fibrillation, was stronger, and its sounds much louder. The mitral systolic murmur, though loud when recumbent, was inaudible when the patient was erect.

In October, the heart was decidedly dilated, with 3 inches of relative dullness in the third left interspace, and 3½ inches of

A CASE IN WHICH THE OCCURRENCE OF GRADUAL CARDIAC ENLARGEMENT INDICATED RETURNING STRENGTH AFTER MYOCARDIAL WEAKNESS

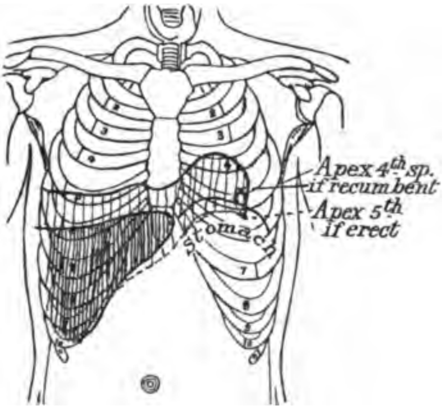


FIG. 1.

Samuel W., aged thirty-eight, August 21, 1913, eight months after rheumatic fever. Mitral regurgitation, but no dilatation or venous plethora because of myocardial weakness. On stairs can only do six or seven steps at ordinary pace.

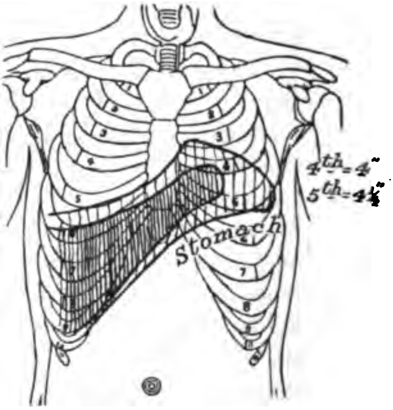


FIG. 2.

Samuel W., September 11, 1913. Heart larger. Sounds louder. Impulse more forcible. Patient less breathless. Can now mount eighteen or twenty steps at ordinary pace. The amount of relative cardiac dullness to the left of the sternum is marked on the diagram.

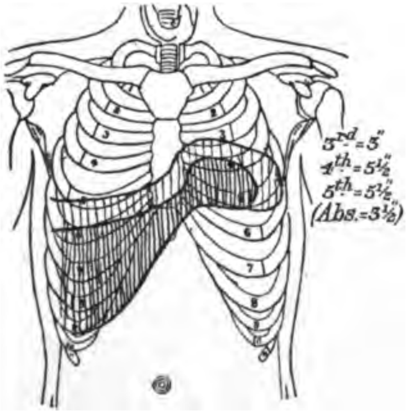


FIG. 3.

Samuel W., October 2, 1913. Heart now considerably dilated, but patient still improving, and less breathless. (Mitral systolic murmur now loud and audible to axilla.)

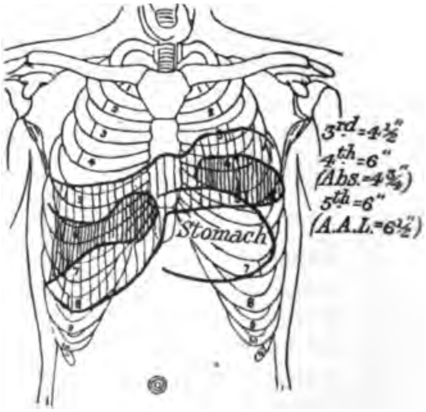


FIG. 4.

Samuel W., November 13, 1913. Heart now greatly dilated (see measurements on diagram), but its action is more vigorous than a month ago; and the patient says he feels stronger, and can walk three miles of rather hilly road in fifty-five minutes.

absolute dullness in the fourth space, and an apex beat 5½ inches from the sternum. The patient was very much stronger, and could walk two miles on the level in an hour without undue breathlessness, but was cautioned not to do so. The mitral systolic murmur was loud and audible into the axilla (see fig. 3).

In November, when seen again, the patient stated that he was stronger and better than at the last visit; but the heart was still more dilated, and the apex was 6 inches from the sternum, nearly in the anterior axillary line, and there was nearly 5 inches of absolute dullness in the fourth interspace. There were now 4½ inches of relative dullness in the third left interspace, but no evidence of dilatation to the right. The heart sounds were now of good loudness, and there was a short rough systolic murmur at the apex, but no auscultatory signs of overaction of the right ventricle (see fig. 4). He could now walk three miles of rather hilly road in fifty-five minutes with only a little breathlessness on the last hill—which was not a steep one.

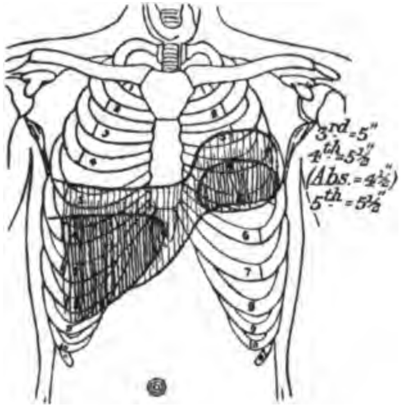


FIG. 5.

Samuel W., December 18, 1913. Dilatation now beginning to lessen (apex half an inch nearer the sternum). Patient continues to improve.

At the next visit the increased strength of the heart began to show itself for the first time by *lessened* instead of by increasing dilatation. The dullness in the third interspace was about the same, but in the fourth and fifth it was about ½ inch less.

The occurrence of dilatation here as the heart gained strength showed its distensibility, and also that the heart muscle had been so weakened by the rheumatism eight months earlier that the thinner part of the ventricle walls gave way as soon as the heart muscle was strong enough to develop an approximately normal amount of intraventricular pressure.

In such a case the reappearance of the cardiac dullness, coupled with increase in the loudness of the heart sounds and the reappearance of the cardiac impulse, can be taken as proof that the strength of the heart is returning. This proof is often of considerable clinical value, for where such a patient is of a nervous temperament, the first few times of getting out of bed often bring about a sense of either weakness, faintness, or actual palpitation. This is apt to cause alarm to the patients and their friends; but I have found that such symptoms can often be ignored if the heart be carefully watched, and that they are of the nature of a habit weakness, and will pass off if the exercise be persevered with. Sometimes it has been necessary almost to *compel* the patient to attempt mild forms of exercise, because of his fear that the discomfort and faintness it induced was the same as that which characterised his attack of true myocardial weakness and which did result in actual fainting. In such cases, where the physical signs show that the heart is not really being injured by the exertion, a change of air and scene will often speedily restore and reassure the patient. The following case illustrates this point: it is also referred to at p. 118, where diagrams of the chest are also given:—

Dr. G., aged twenty-six, had had an attack of diphtheria six weeks ago. There had been no paralysis, but the patient had had troublesome heart attacks ever since and had been confined to bed. The attack consisted of intermission of the pulse with a sense of heart weakness and faintness, making the patient afraid to move.

On examining her it was found that the pulse was small and weak, the veins of the neck were empty, and there was no evidence of any venous engorgement of the liver: on the contrary, all signs pointed to emptiness of the heart and veins.

There was no cardiac impulse to be seen or felt anywhere, and there was no cardiac dullness to be detected, for the full gastric resonance came as high as the lower border of the fourth rib, and was here met by the full lung resonance, showing a very high diaphragm. The liver dullness, too, was much smaller than normal. The heart sounds were free from murmurs, but were very weak and wanting in vigour. A fortnight later, it was found that there were some signs that the heart was stronger. There was a trace of relative cardiac dullness in the fourth interspace internal to the nipple line, though there was still some gastric resonance in this same interspace; there was also a trace of cardiac pulsation to be detected here. On auscultation, there was more vigour about the heart sounds: they were not quite so weak and short, the second sound was moderately loud at the apex, and there was a faint systolic murmur over the right ventricle in the fourth interspace near the sternum. The liver dullness was small. Its absolute dullness began about the normal level in the sixth interspace, but it did not reach to within 1½ inch of the costal arch, nor did it come internal to the right vertical nipple line. The relative liver dullness began above at about the normal level, but did not quite reach down to the costal arch, and there was only a trace of liver dullness in the middle line at the base of the sternum (*vide* figs. 16, 17, 18; and 118).

The patient progressed very slowly; but when seen two weeks later, there was well-marked cardiac pulsation in the fourth interspace, and a trace of pulsation in the third, and the apex was under the fifth rib and was just to be felt in the upper part of the fifth interspace in the nipple line.

At the sternal end of the fourth interspace, the cardiac dullness was nearly absolute.

The liver dullness had not increased in size, but was, in fact, a trifle less; for its absolute dullness did not reach to within 2 inches of the costal arch.

After another week or two, as the heart continued to gain strength, the patient was allowed out of bed; but after getting up a few times, the question arose as to the wisdom of doing so, for it not only produced a feeling of faintness and exhaustion, but also a certain amount of palpitation which somewhat alarmed the patient. However, a careful examination of the heart showed that it was evidently stronger, the sounds were louder, and there were no signs of any dilatation, and the patient was advised to continue to get up and take a gradually increasing amount of exercise. The result was entirely satisfactory. This case is again referred to, in connection with palpitation as a cardiac habit, at p. 118, and figures showing the changes in the size of the heart and liver are there given.

EARLY DIAGNOSIS OF HEART FAILURE IN THE RELATIVELY RIGID HEART OF LATER LIFE

SUMMARY

INTRODUCTORY.

- (i) Modifications in the signs and symptoms of heart failure which result from the increasing rigidity of the fibrous tissues which characterises advancing life.
    1. As regards the body generally.
    2. As regards the heart and pericardium.
- (ii) Modifications in the signs and symptoms of heart failure due to the arterial changes of later life.
    1. Rise in blood pressure.
    2. Disease of coronary arteries.

I. DISTENSIBILITY OF THE HEART IN LATER LIFE.

II. EARLY DIAGNOSIS OF HEART FAILURE WITH ENLARGEMENT IN RELATIVELY RIGID HEARTS.

III. HEART FAILURE WITHOUT ENLARGEMENT IN STRONG OR ONLY MODERATELY WEAK HEARTS OF CONSIDERABLE RIGIDITY.

IV. HEART FAILURE WITHOUT ENLARGEMENT IN DECIDEDLY WEAK AND RELATIVELY RIGID HEARTS.

SYMPTOMS: Dyspnœa, faintness, pain, palpitation, physical signs.

Illustrative cases of cardiac failure in weak and relatively rigid hearts.

V. EARLY DIAGNOSIS OF MUSCULAR FAILURE OF THE HEART IN LATER LIFE IN CASES OF VALVULAR DISEASE.

VI. SHORT NOTES UPON SOME OF THE DIAGNOSTIC DIFFICULTIES WHICH SURROUND THE RECOGNITION OF HEART FAILURE IN LATER LIFE, AS REGARDS—

1. Dyspnœa.
2. Palpitation.
3. Faintness.
4. Anginous pain.
5. Venous phenomena.
6. The recognition of arterial resistance as a factor in cardiac failure.
7. The recognition of rigidity of the heart wall as a factor in cardiac failure.
8. Difficulties in connection with the physical examination of the heart.
9. Difficulties due to one ventricle being weak while the other is strong (with illustrative cases).
10. Summary of various types of difficulty.

INTRODUCTORY

This section on the early diagnosis of heart failure in later life must be commenced with an apology, because the subject, in the present imperfect state of our knowledge, is a very difficult one to treat concisely and with scientific method.

The fact that many of the physical evidences of heart failure, upon which we rely for purposes of diagnosis in early life, are less marked or absent in later life, and that we often have to diagnose heart failure *because* of the absence of such physical signs, does not help to make the subject an easy one to deal with. Again, in later life many of the evidences of heart failure are capable of bearing exactly opposite interpretations. For instance, fullness of the veins of the neck may be a sign of the failure of a moderately strong heart, or its appearance may be the sign that a weak heart is gaining strength. Again, a lessening in the degree of fullness of the neck veins may be the sign that a strong heart is gaining strength, or that a weak heart is losing strength (*vide* p. 105 below). Diminution in the size of the right ventricle may mean return of strength in a moderately strong heart, or dangerous weakness in a weak heart. Again, another difficulty which has to be faced is that the absence of well-marked *signs* in later life necessitates a far greater reliance upon *symptoms* for the diagnosis of heart failure: and symptons are—especially in adult and later life—very unreliable, because of the frequency with which symptoms of true cardiac failure are simulated by symptoms which arise merely from reflex disturbances, and vice versa.

In order to try to bring some method into the treatment of this complex subject, it is proposed first to discuss the main differences between the heart in later and early life and the main points upon which reliance can be placed, and then to take up the diagnostic difficulties in connection with the symptoms and signs of heart failure, illustrating the various points by giving actual clinical cases.

In summarising the various points upon which we have to rely for the early diagnosis of heart failure in later life, we find that there are distinct differences as compared with those already described as characterising heart failure in early life, and the differences depend mainly upon two characteristics of mature and later years—namely:—

1. The tendency which the fibrous tissues of the body generally have to consolidate and become more rigid as life advances, and thus to modify the signs and symptoms of heart failure; and

2. The tendency to abnormalities of the arteries, either of the nature of spasmodic contraction and rigidity—such as is associated with faulty elimination of waste products—or abnormalities due to degenerative changes in them.

To take up the first group.

I. CHANGES DUE TO INCREASED RIGIDITY OF FIBROUS TISSUES.—1. So far as the general tissues of the body are concerned, the only modifications will be in those signs of heart failure which depend upon variation in the position or size of organs.

Thus it is not to be expected that in later life alterations in the volume of the intrathoracic contents will result in alterations in the level of the diaphragm as readily as is the case when the fibrous tissues are elastic, earlier in life. More especially is this the case with regard to the right half of the diaphragm, and in later life it is not usual to find any marked rise in the upper level of the liver dullness in myocardial weakness, however severe.

Rise of the left half is, however, a valuable sign, although it be less extreme than earlier in life.

So far as alteration in the size of the liver dullness is concerned, the more rigid tissues of later life will not show such extreme changes when the organ is underfilled with blood as was the case earlier in life.

2. Changes in the Heart and Pericardium.—The chief bearings of these changes upon the early diagnosis of heart failure in later life are as follows :—

(1) Where heart failure is accompanied by enlargement, although the *type* of dilatation will be the same as that occurring in adult life (p. 31), the *degree* of enlargement will be less, and the greater the rigidity the less will the amount of dilatation be for any given amount of distensile force.

(2) When the degree of rigidity of the heart is very considerable there may be no yielding at all of the heart wall, in spite of the heart being of considerable muscular strength.

Take, for instance, a case of overstrain in a heart of good muscular strength with a moderately distensible wall. In this case the rise in intraventricular pressure necessary for the carrying on of the circulation may be in excess of the resisting power of the heart wall, and the heart then dilates. Take, however, another heart of the same muscular strength and facing the same amount of overstrain, but in which the fibrous rigidity of the wall is so great that it can withstand the increased intraventricular pressure.

Although in the two cases the heart muscle is strained to the same extent by meeting the increased resistance it has to face, there will, in the one case, be heart failure with enlargement, and in the other, heart failure without enlargement.

This is a point upon which stress must be laid. In studying heart failure in later life, the muscular strength of the heart must not be confused with the fibrous strength of the heart wall, and the two must be considered separately. A heart that dilates in the face of overstrain is, in later life, often stronger muscularly than one that does not dilate.

Therefore, in later life, we must recognise a fresh group—namely, cases where there is heart failure without enlargement in moderately strong hearts.

(3) Rigidity of the heart wall will interfere more or less completely with the development of compensatory diminution in the size of the heart in extreme myocardial weakness.

II. Modifications in the Signs and Symptoms of Heart Failure due to the ARTERIAL CHANGES of later life.

The second of the two main changes that we have spoken of must now be taken up—namely, he changes in the arteries.

1. RISE IN BLOOD PRESSURE due to reflex or organic narrowing of the peripheral arteries.—The diagnosis of early cardiac failure is rendered more complicated in later life by the greater frequency with which changes in the blood pressure occur, for, although cases showing high blood pressure are sometimes found in adolescence and early adult life, they are by no means common, whereas in later life they are extremely frequent.

Therefore, in an adult, in estimating the amount of work the heart is doing, we not only take into account the energy necessary for the amount of exertion the patient is making, but we must also be careful to estimate as nearly as possible the extra work that may be thrown upon the heart by the interference with the circulation which abnormal rigidity of the smaller arteries brings about. The uncertainties which are apt to make this, at times, far from easy are dealt with later on when considering 'Diagnostic Difficulties in Later Life.' The different causes of high blood pressure do not come within the scope of our subject, and are, moreover, dealt with fully enough in medical literature.

Another condition which may greatly complicate the diagnosis of heart failure is rigidity of the first part of the aorta as a result of atheroma or of a more rare inflammatory affection—namely, chronic mediastinitis. Considering that the first part of the aorta (aided by the elasticity of the arteries) acts as an expansion chamber, allowing the heart to keep up a continuous flow of blood, although it merely gives a short sharp stroke, interference with the elasticity of this portion of the aorta will necessitate on the part of the ventricle a long-sustained contraction in order properly to drive the blood into the peripheral vessels, and this may readily lead to cardiac failure. Its presence may cause uncertainty in diagnosis, for it may not be easy to understand the reason for a strong sustained pulse in a dilated and hypertrophied heart with no apparent cause for failure.

2. Disease Affecting the Coronary Arteries.—The liability of the heart in later life to suffer from malnutrition, owing to disease of the coronary arteries, forms one of the most striking differences between heart failure in early and in later life, and greatly adds to the difficulty in recognising cardiac failure. When speaking of the cause of heart failure with small heart in adolescence, attention was drawn to the fact that the main agencies which produced myocardial weakness were general ones—such as the toxæmia of influenza, diphtheria, rheumatism, &c. These agencies, in spite of the fact that there may be small localised inflammatory foci in the heart muscles (such as have been demonstrated in rheumatic myocardial weakness by many observers), rarely, if ever, seem to involve one ventricle more than the other, and clinical symptoms of *general* muscular failure therefore result.

When dealing, however, with the adult heart the commonest causes of muscular weakness are circulatory in origin—due to interference with the circulation in the coronary arteries by atheroma. Under such circumstances it is by no means uncommon for the arterial supply of one ventricle to be interfered with to a greater extent than that of the other.

Therefore, we may meet with hearts where one ventricle is moderately strong, while the other one is very weak; and we may occasionally be surprised to find a patient dying of breathlessness of cardiac origin, in spite of the fact that the pulse is fairly good and strong; for this is what occurs where the coronary supply to the right heart is greatly interfered with, while that of the left ventricle is fairly good. Such a case is described on p. 154. Where, on the other hand, the left ventricle is the weaker, the diagnosis may be more easy if fainting or marked weakness occur; but it may be quite as difficult as in the case of the right ventricle—for example, in the cases where a patient dies suddenly from fatty degeneration without his medical attendant being aware that there is any dangerous weakness of the heart.

This subject will be further dealt with and illustrative cases given in the Essay on 'Diagnostic Difficulties in the Heart Failure of Later Life.'

DISTENSIBILITY OF THE HEART IN LATER LIFE

The distensibility of the heart in later life must now be discussed in its relation to the early diagnosis of cardiac failure.

The amount of distensibility possessed by the heart plays, in later life, an even more important part as regards the diagnosis and prognosis of heart failure than it does in early life; but its amount is much less easily ascertained, and for this reason, that, in adolescence, owing to the softness of its tissues, the heart changes its form and size easily, giving positive clinical evidence of its distensibility, whereas in later life its maturer tissues yield much less easily, and thus the signs of its lessened distensibility are mainly negative.

But before going further with the subject, this whole question as to the rigidity of the fibrous tissues increasing with advancing age must be discussed in a little more detail, because I am not aware that it is accepted, so far as the heart is concerned, as a proved fact; and the anatomist may object that there is not enough true fibrous tissue in the heart wall to render probable such a change as that here suggested.

In the first place, this proposition as to an increase in the rigidity of the fibrous tissues is certainly accepted, so far as the tissues of the body generally are concerned, and it will not be disputed that one of the main differences between early and later life lies in the condition of the fibrous tissues, which in early life are relatively soft and distensible.

The body is, to use a familiar phrase, more supple in early life. With advancing years, the tendency of the fibrous tissues is to become less and less distensible, the joints get stiffer and less easily adaptable to altered uses—such as the learning of new gymnastic exercises, &c. Dislocation of the joints, too, is less likely to occur than in early life, owing to the greater rigidity of their fibrous supports. This change, if it also involved the cardiac tissues, would account for many of the differences which exist between the heart in early and in later life. The clinical evidence in favour of such a hardening of the tissues of the heart sometimes taking place in later life, is so strong as almost to amount to proof—in spite of the fact that anatomists will tell us that there are practically no fibrous elements present in the tissues of the heart wall, and that, therefore, a change which involves the fibrous tissues of the body generally cannot be expected to affect the heart wall; but if this is really so, then the change which we often detect clinically in the heart of advanced life must be due to some corresponding change taking place in the muscular fibres themselves.

Even if this theory of the heart wall itself partaking in the increasing rigidity, which age induces in the fibrous tissues of the body, be ultimately proved to be wrong, and some other explanation be found for the phenomena here described, the writer asks that for the present this theory be allowed to stand provisionally, if only as a working hypothesis, because it suffices so well to explain and bring into line so many of the differences which exist between the heart in early and in later life, and helps to supply a means of, to some extent, bringing order into a subject which, in the present state of our knowledge, is at best somewhat chaotic. It is asked, therefore, that this limitation be accepted, in connection with the dogmatic statements in the following pages, by those who cannot agree wholly with the writer of them.

Again, in order to avoid misunderstanding, it is desirable to define clearly the sense in which the word 'rigidity' is used in this connection; for the heart does not, in the absolute sense of the term, possess any rigidity at all. It is used here merely in a relative sense—for want of a better word—and when used must be understood to imply lessened distensibility—namely, an increase in the amount of the opposition which the cardiac tissues (including the pericardium) can offer to any force tending to cause an alteration in the shape or size of the heart.

The word 'distensible' has been used to describe the condition of the heart in the first two or three decades of life; for it is easily distended, the soft and still developing fibrous tissue of the pericardial sac having but little controlling influence upon any tendency the heart may have to become enlarged. As the fibrous tissues of the pericardium get stronger with age, they will exert more control over the distensibility of the heart, and to this control is, in all probability, added another agency—namely, a certain amount of stiffness or added firmness, which the tissues of the heart itself often seem to gain as life advances.

This gradual change has already been spoken of in connection with the altered type of dilatation, which is seen in adult as compared with early life. This same lessened tendency to enlargement (due to changes both in the heart wall and in the pericardium) goes on increasing with the increasing rigidity of the tissues (which is apt to characterise advancing age), until in later life the heart's walls may so have lost their distensibility that a degree of overstrain which would have been certain to cause dilatation earlier in life has now no effect in enlarging the heart.

Shortly to describe this change in the character of the heart, it may be called 'the gaining of an increasing degree of rigidity as age advances,' and the less dilatable heart of later life may be called 'the relatively rigid,' or simply 'the rigid' heart of advanced life.

But this increasing rigidity does not only affect the ease with which the heart is dilated. There is every reason to believe that the heart walls are less easily compressed and deformed, by the contractions of its own muscles, in later life than it is in early life. In other words: that the increased hardening of the tissues with age involves, not only the two layers of the pericardium, but also the wall of the heart itself; and that in later life the heart will, when diminished in bulk by contraction of its muscles, tend to return to its original size with greater force than it does in early life. Therefore, after the ventricle has been diminished in size during the act of contraction, it will, as soon as the contractile force ceases, tend immediately to return to its original size, and in so doing, it will suddenly increase the capacity of its ventricle and cause a certain amount of suction to be developed within it.

Now, here again, the word 'rigid' is not very suitable, because a rigid body is not elastic; whereas the slight hardening of the cardiac tissues, which is here spoken of as relative rigidity, does impart to them some mechanical elasticity, and so gives the heart the power of mechanical aspiration, as well as the muscular aspiration already described.

With these words of explanation and apology as to the use of the word 'rigidity,' and also as to the theory here propounded as to the increasing resilience of the heart in later life being brought about by the hardening and consolidation of its tissues, we will proceed to deal with the bearings of this theory upon the symptoms and signs of the more extreme cases of cardiac weakness in later life.

Results of increased rigidity of its tissues so far as THE HEART ITSELF is concerned.—In the first place the increase in firmness, which we are assuming that age is likely to impart to the cardiac tissues, will—as already pointed out—lessen both the readiness with which the wall of the heart dilates when exposed to abnormally great intraventricular pressures, and also make it resist other types of deformation as well. We shall not, therefore, expect to find in later life the great diminution in the size of the heart of which we have already spoken when dealing with extreme cardiac weakness in early life.

But this increased resistance to change of form will affect the diminution in the size of the full heart in another way—namely, by interfering to some extent with the compensatory mechanism whereby the lessening in size of the heart is brought about. When the distensible heart of an adolescent has contracted, its soft fibrous elements, having but little resiliency, can take but little part in restoring the ventricle to its original shape, and this has to be done by muscular expansion alone. In later life, however, the tissues of the heart being more rigid, there will be more inherent tendency—apart from muscular expansion—for the ventricle to return to its full size as soon as contraction is over. In other words: this quality of the tissues will ensure the heart having a certain amount of mechanical elastic recoil in addition to its power of muscular expansion.

When dealing with the mechanism which ensured the underfilling of the heart in extreme muscular weakness, we pointed out that expansion being a less effective action than contraction, it might reasonably be expected that it would diminish in effectiveness to at least as great an extent as was the case with contraction, and that therefore in a heart which filled itself solely by its own muscular expansion muscular weakness might be expected to interfere with the *filling* quite as much as with the *emptying* of the ventricle, and that in all probability defective filling would be the first of the two phenomena to develop.

Now, in later life the possession of the mechanical resilience of which we are speaking must, to some extent, interfere with this compensatory mechanism by giving the heart the power of mechanical aspiration in addition to that due to muscular expansion. We can, therefore, say that there is good reason to expect that in later life there will be more complete *filling* of a muscularly weak ventricle than would be the case in early life.

In other words : in later life, we must not expect to find clinically such marked diminution of the cardiac dullness in cases of myocardial weakness as is the case in early life.

The firmness of the fibrous tissues in maturity may help to bring about this same result in another way, for some of the apparent diminution in the size of the cardiac area, when the patient lies on his back, may be due to the underfilled heart falling away from the chest wall to a greater extent than normal. This also would be, in some measure, less in extent when the tissues of the heart and the mediastinum were firm than when they were soft.

We find, therefore, that while smallness of the full heart (from underfilling) is an almost certain accompaniment of extreme muscular weakness in adolescence and early life, in middle and later life, on the other hand, this diminution in size cannot be expected to occur to the same extent, and may even be absent altogether, so far as our powers of observation are concerned.

It may, therefore, I think, be stated that just as the degree of distensibility of a heart that is only moderately weak is shown by the type and the extent of its *enlargement*, so, on the other hand, the degree of distensibility of an extremely weak heart is shown by the extent of its *diminution* in size. The more distensible the heart the greater will be the diminution in size of the heart when full, and the less the distensibility, the less will be that diminution.

Although the possession of mechanical elastic recoil is here only inferred from the clinical phenomena which the heart exhibits, it is possible that the difference between the heart of early and later life in this respect may be demonstrable by experiment in the post-mortem room, and it would certainly be an interesting field for research. It is not, however, at all certain that it would be demonstrable, because of the presence of rigor mortis—for the rigidity of the muscles would far exceed any elasticity which the scanty fibrous elements of the heart might be expected to be capable of. Moreover, it is possible that the formed elements of the muscular fibres themselves may play an important part in producing the phenomenon which we are now discussing. If this be so, it is not easy to see how after death this quality could be demonstrable.

The possession of some measure of mechanical elastic recoil may, therefore, be taken as an important characteristic differentiating the heart of middle and later life from that of early life, and one upon which some of the many clinical differences between hearts at those two periods of life depend.

More stress must not be laid upon this point than it will rightly bear, for it must be remembered that anatomically there appears little or no fibrous tissue in the heart wall, and also if mechanical resilience be granted, the force which brings it into play is, after all, the contractile power of the heart wall, and that when a heart is weak its more feeble systolic squeeze will be followed by only a feeble elastic rebound. Therefore, unless the heart be very rigid, there will be some compensatory diminution in its fullness, and the difference between the smallness of the heart in the distensible and the relatively rigid heart will only be one of degree.

In the case, on the other hand, of a heart which combines considerable weakness with a considerable degree of rigidity, it is probable that the weak muscle will be unable fully to compress the relatively inelastic heart walls, and that there will be incomplete emptying of the heart at each systole.

If this be so, the effect upon the general circulation will be the same as when an underfilled ventricle completely empties itself; for in both cases a portion only of the normal amount will be discharged into the arteries at each contraction, and, in this type of heart, the weaker the heart muscle the less perfect will be the emptying of the ventricle during its contraction.

Therefore, in this case also, we can estimate clinically, by the extent of the deficiency in the amount of blood in active circulation, the extent of the muscular weakness of the heart.

It has been already pointed out that in adolescence, owing to its power of aspiration failing before its power of expulsion, the risk of sudden death from asystole is reduced to a minimum; whereas in later life the possession of mechanical resilience deprives the heart of this safeguard, and therefore sudden death, due to the left ventricle receiving a larger amount of blood than it has power to expel, is by no means an uncommon accident in cases of extreme myocardial weakness.

Further, in cases where death does not follow in such a case, anginous pain is caused by the efforts of the ventricle to exert a force beyond what it is readily capable of doing. This is, I believe, one explanation of the fact that anginous pain in simple myocardial weakness is rare in early life, but by no means uncommon in later life.

In valvular disease where, in consequence of valvular incompetence and the resulting regurgitation, over-filling of the left ventricle can occur, anginous pain is frequent in early, just as in later, life.

Before leaving this subject of distensibility of the heart in later life, it is necessary to draw attention to the fact, already pointed out, that there is great variation in the amount of distensibility possessed by the heart in later life, just as was shown to be the case in early adult life.

For, while in some patients the heart appears to be so rigid that it hardly seems to dilate at all under circumstances that would certainly have caused marked dilatation earlier in life, we may, on the other hand, sometimes meet with patients in whom at the age of sixty the heart appears to dilate nearly as readily and in the same manner as it would do in a patient only thirty years of age.

It need not be pointed out that, so far as the general fibrous tissues of the body are concerned, we expect such variations; for one man will remain lissom and active till he is over sixty, whereas another will become stiff and aged before that time.

It may be of interest to give some details of two elderly ladies, in whom the heart was distensible and 'young,' for comparison with two other cases, which are given at pp. 87 and 89, in whom the heart was rigid and old.

The patient was a lady aged sixty-two, who was suffering from tachycardia, which ultimately showed signs of becoming a true exophthalmic goitre, and was cured by a partial excision of the thyroid.

When seen by the writer, her heart was much dilated and was beating 140 to the minute. This was in part due to the presence of the consulting physician, but its rate never fell below 100.

Her urgent cardiac symptoms dated some months back, and were brought on after an 'influenzal' attack by a series of somewhat trivial causes—showing that the heart was in a very over-sensitive condition.

Firstly, slight over-exertion brought on an attack of asthma followed by cardiac dilatation and a mitral murmur. On recovery, too long a drive caused a return of the symptoms and a pulse rate of 140 with occasional extra systoles.

The cardiac action was very excited, and the sounds so loud that they were audible when the stethoscope was one-third of an inch away from the chest wall.

The apex beat was in the fifth interspace, about midway between the left nipple line and the anterior axillary line, and there was enlargement of the cardiac area upwards and to the left. There was no dilatation to the right or downwards, and the right lung crossed over the middle line and was well to the left of the sternum in the fourth and fifth interspaces, and during full expiration the edge of the right lung only retreated as far as the left border of the sternum in the fourth left interspace—thus showing either displacement of the heart towards the left or diminution in the size of the right ventricle.

The dilatation was wholly outwards and to the left, and the relative dullness extended well beyond the left nipple line in both the third and fourth interspaces.

The only murmur was a faint mitral one, and there was an extra sound audible over the right ventricle which was probably a third sound; but as the heart was beating 140 to the minute, the proper place for the third sound would be less than one-tenth of a second before the succeeding contraction, and therefore it would, as was the case here, appear like a reduplication of the first sound, although with such a limited area of audition as to preclude the possibility of this explanation.

Adolescent Type of Dilatation
occurring in Late Adult Life

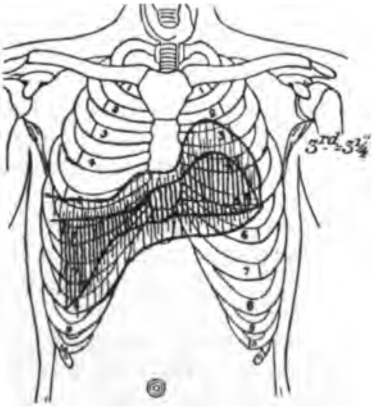


Fig. 6.

Mrs. S., aged fifty-two. Shows upward dilatation of the right ventricle, the result of overstrain.

The striking feature about this case was the fact that the mode of dilatation of the heart was that of an adolescent and not of a woman of sixty-two.

There was in this case no evidence of any dilatation of the left ventricle other than the outward displacement of the apex; for the cardiac sounds were heard less loudly than normal over the situation of the left ventricle and the left auricle, posteriorly.

The peculiar shape of the dilated heart was probably due, as is the case in anæmic dilatation, to two factors: firstly, the distensibility of the anterior wall of the right ventricle; and secondly, to the occurrence of a dilatation of the right ventricle, combined with a left ventricle of normal or less than normal size.

The second case is that of—

A lady aged fifty-two, who came to see me for heart weakness, which showed itself not only by some breathlessness on exertion but also a great sense of feebleness and want of energy.

There was no evidence of œdema of the ankles or cyanosis.

The heart was found to be markedly dilated upwards and to the left, just as occurs in overstrain in adolescence.

There was some relative dullness and pulsation in the second left interspace and well-marked pulsation in the third interspace with relative dullness extending some 3½ inches from the sternum. The absolute dullness just reached into the third interspace. The apex was high in the fifth interspace well external to the nipple line. The liver dullness was small. There was no murmur at the apex (see fig. 6).

If the diagram here given is compared with figs. 48, 99, the similarity of the dilatation here to that occurring in adolescence will be evident. The patient did not, however, recover with the rapidity of an adolescent, and needed prolonged rest and care.

Such cases are, however, rare, and as a rule the manner and the amount of the dilatation seem to point to the fact that the rigidity of the heart wall increases steadily as age advances.

Early Diagnosis of Heart Failure with Enlargement in Relatively Rigid Hearts

In the relatively rigid heart of advanced life, when dilatation occurs, we find it to be of the same type as that already described at p. 31, as occurring in adult life. The principles, therefore, governing its early diagnosis are the same as those there given. It must, however, be remembered that owing to the possibility in later life of the cardiac tissues being less distensible it must not be concluded that a slight degree of enlargement at this time of life means only the slight amount of overstrain that it would mean in early adult life.

Considering the degree in which rigidity of the pericardium and of the walls of the heart can limit the heart's distensibility an endeavour must be made to estimate what the degree of distensiblity is, before any estimate can be made of the amount of overstrain which any particular amount of dilatation represents.

Some help in this estimation may be sometimes gained by noticing the general build and habit of the patient, but the only reliable guide in judging of the amount of overstrain is obtained from noting the strength of the right ventricular beat as shown by the heart sounds and the condition of the veins, and especially by study of the pulse in the jugular bulb. If we find distinct evidences of considerable overaction of the right ventricle, combined with but slight enlargement of the heart, we must lay stress upon the overaction which we can notice, and conclude that the absence of a more noticeable amount of dilatation must be due to rigidity of the heart walls. But if we find a moderately acting right heart combined with slight enlargement, we are at once at a loss, so far as the estimation of the amount of its failure is concerned; for the case may be one of rigid heart with a muscularly weak right ventricle, and in a condition of severe overstrain, in spite of the slightness of the venous phenomena.

This is but an instance of the caution necessary in later life as regards reliance upon negative phenomena.

A phenomenon that is certainly abnormal is of value, but an apparently normal one has not the same value.

In later life, as under many other circumstances, a reliable diagnosis cannot be made till all the facts of the case have been considered and their due value apportioned to them in the light of all the phenomena observed.

HEART FAILURE WITHOUT ENLARGEMENT IN STRONG OR ONLY MODERATELY WEAK HEARTS OF CONSIDERABLE RIGIDITY

As has already been pointed out, the development of the marked 'rigidity' which we occasionally find in the heart of later life necessitates the formation of a fresh group in addition to the two groups of 'strong heart with enlargement,' and 'weak heart without enlargement,' which included practically all cases of cardiac failure in early life.

In this group, we shall deal with cases where the pericardium and heart wall have so lost their distensibility (as a result of senile or other changes) that a rise of intraventricular pressure which, earlier in life, would have been certain to cause dilatation of the heart, is now powerless to do so—so far at least as our powers of clinical observation are concerned.

The difficulties in the way of a correct diagnosis of heart failure are in later life greatly added to by the recognition of the fact that we must not (owing to the rigidity of the heart) expect dilatation under circumstances that would certainly cause it earlier in life.

This also must limit the number of simple cases with cardiac dilatation which we are likely to meet with, while it adds to the number of the more complex and difficult cases where heart failure is unaccompanied by enlargement.

No hard-and-fast line can be drawn between these cases and those in the last group; for the strength of which we are assuming the heart is possessed is adequate to dilate a normal heart wall though not an unduly rigid one, and under some circumstances there may quite possibly be some dilatation of ventricles or auricles although there be no clinical evidence of it. Practically, this group belongs to the preceding one of heart failure with enlargement, but for purposes of clinical diagnosis it is convenient to separate it.

If we regard the 'auriculo-venous reservoir' (*vide* p. 132) as part of the heart proper, we should have, even in these cases, to speak of cardiac dilatation being present.

The first of the following cases is one of these border-line ones, and will serve to illustrate this group, though it was not possible absolutely to exclude the presence of some cardiac dilatation. In the second case here recorded, there was certainly no cardiac enlargement.

The diagnosis of this group of cases is well illustrated by the accounts here given.

The first case is that of an elderly man with well-marked arcus senilis and atheromatous radial and temporal arteries, who was admitted to hospital suffering from breathlessness and a certain amount of dropsy of the ankles. The heart was very slightly, if at all, enlarged.

There was, however, well-marked venous pulsation in the neck, showing that, although the muscular strength of the ventricles was not great enough to cause material dilatation of their cavities, that of the right was sufficient to keep the veins fully distended and pulsating.

There was, moreover, a loud systolic murmur heard in the second and third right interspaces, and well conducted up into the vessels of the neck. This murmur, by its character—its area of audition and its direction of conduction—very closely resembled an aortic systolic murmur. This case is impressed upon the writer's memory by the fact that his house-physician, who was a keen observer and a young man, fearlessly opened the following discussion in answer to his chief's suggestion that this murmur was produced in the superior cava and was a sign of tricuspid regurgitation. He said: 'Why make such a foolishly fanciful diagnosis? Here we have an oldish man with well-marked arcus senilis and atheromatous arteries, and just the patient in whom one would suspect atheromatous roughening of the aorta and an aortic systolic murmur.' To this the answer was: 'I am sorry, but my fingers tell me that these are dilated veins in which the murmur is heard, and my ears tell me that this murmur is very loud over the second interspace and not nearly so loud in the arteries of the neck as an arterial murmur would be. If the blood in the aorta were vibrating enough to make a loud murmur audible through the inch of lung tissue between it and the chest wall, that same blood, a fraction of a second later, when it reaches the carotid arteries, must be still vibrating; and with a stethoscope applied directly on to the artery the murmur would be better heard than this one is. Moreover,' I added, 'this murmur is well heard low down in the second, and also in the third, interspace in a manner that an aortic systolic murmur is not.'

These facts did not satisfy the young observer, and, although a few days' rest in bed—a little strophanthus and salicine—sufficed entirely to remove the dropsy, the over-distension of the veins, and the murmur, when the notes of the case were put away ready for binding, I noticed that the diagnosis was entered up as 'aortic systolic murmur.'

Such a case as this shows that in a relatively rigid heart we cannot depend—for the early diagnosis of heart failure—upon changes in either the right or left ventricle as signs of dilatation, or upon the presence of systolic murmurs—although the latter, when present, are of great value—but we may have to depend entirely upon phenomena due to over-distension of the great veins. This case is also referred to at p. 222.

The following is another border-line case in which a degree of muscular weakness as well as rigidity of the heart wall brings it nearer still to the cases of heart failure without enlargement, which will be next dealt with.

The patient was a stout, red-faced man, aged forty-four, whose work as an agent involved a good deal of cycling. For many months he had noticed an increasing breathlessness, and he had been unable to do much cycling for more than a year. Within the last few weeks, his breathlessness had been so troublesome that he came to the hospital and was admitted. There had been no swelling of the ankles and no fainting attacks. On admission, the most evident signs of cardiac failure were fullness of the veins of the neck with well-marked pulsation in them as far up as the angle of the jaw; but the pulsation, though greater in extent, was less forcible than in the last case. The pulse was small in volume and wanting in vigour, the tension was rather high and the blood pressure, as tested by the sphygmomanometer, was 160 mm. of mercury.

As regards the heart. Instead of finding distinct dilatation, and perhaps a tricuspid murmur, as might be expected under such circumstances, percussion showed the cardiac area to be normal, and on palpation and inspection the cardiac action and impulse were found to be more feeble than normal.

Auscultation confirmed this; for the heart sounds were decidedly weak, and those at the apex were short and sharp, suggesting feebleness, and there was also a very faint systolic murmur present. The right ventricular sounds, while rather better than those at the apex, were not as loud as normal, and there was no murmur. As regards the lungs, there was no emphysema, but there was a good deal of bronchial wheeze, suggestive of bronchial catarrh with some bronchial spasm.

Here, again, we were dealing with a case of heart failure without manifest dilatation, although some dilatation of the right auricle and possibly of the right ventricle might nevertheless exist, and the mitral systolic murmur suggested left ventricular dilatation. The explanation of such a case is probably as follows: The man had been suffering more or less for some time from a gradually rising blood pressure—the result probably of failing kidney action or an injudicious dietary, and the nutrition of the heart had not been sufficient (owing to faulty coronary arteries) to enable it to keep pace with the extra work thus thrown upon it. Had it done so, there would have been a measure of hypertrophy with heart sounds louder than normal and a murmur due to tricuspid regurgitation. The final cause of his breakdown was doubtless his bronchial trouble. The fact of having a heart of normal size and weak action, in face of a blood pressure of 160 mm. of mercury, suggests myocardial degeneration from malnutrition (see p. 144).

The distension and pulsation of the veins showed that the right ventricle was relatively strong and was endeavouring to compensate for the defective action of the left heart.

As regards the treatment: the bronchial catarrh and spasm, though only slight, had to be dealt with, and also small doses given of such a drug as strophanthus which, while not increasing arterial contraction, does increase the vigour of the cardiac action, and so tends to improve the cardiac nutrition. Digitalis would not be indicated. A very important part of the treatment of such a case is the lessening of the peripheral resistance by means of drugs such as the salicylates. In this case, however, the prognosis was not hopeful as in the last one, owing to the myocardial weakness. This was shown by his rate of progress; for, although the heart sounds gained in vigour after three weeks' rest in bed, combined with the treatment described, the man progressed but slowly.

These two cases will suffice to show the points upon which the early diagnosis of heart failure in relatively rigid hearts must depend.

Breathlessness, combined with fullness and pulsation of the veins are the first signs. In a moderately strong heart there will be a systolic murmur due to tricuspid regurgitation: either a true tricuspid systolic with or without a superior vena cava murmur, or else the latter murmur alone, and there will probably be a mitral systolic murmur as well. In a weaker heart the right ventricle, while strong enough to cause some regurgitation into the neck veins, may yet be unable to develop enough pressure to give rise to a murmur at the tricuspid orifice or in the superior vena cava.

If the heart were weaker still, it would come into the group (now to be discussed) of ‘ Heart Failure without Enlargement in Weak Hearts.’

HEART FAILURE WITHOUT ENLARGEMENT IN DECIDEDLY WEAK AND RELATIVELY RIGID HEARTS

The most difficult of all the groups into which we have divided cases of heart failure in later life has now to be discussed—namely, the group which contains cases where the myocardium is too weak to cause any dilatation of even a normally distensible heart wall.

After what has already been said as to the variations in the distensibility of the heart, it must not be forgotten that even in later life an exceptional case may be met with to which the description already given (p. 56) of the muscularly weak heart of adolescence would apply. But in the majority of the cases of this type met with in later life the signs and symptoms are modified by the increase in the firmness and resiliency of the heart wall which has already been fully discussed.

In this place, we shall simply take up the main diagnostic points upon which we must rely for the recognition of heart failure in such cases, together with one or two illustrative cases, and postpone to p. 112 the discussion of the many and serious complexities and uncertainties which we are only too apt to meet when studying the signs and symptoms of heart failure in later life.

First, as to symptoms of heart failure. As has already been pointed out, the negative character of much of the evidence in this class of case renders it more necessary than in early life to rely upon symptoms of cardiac failure. Owing to the uncertainty which often attaches to the symptoms of any ailment, we must be specially careful in our analysis of them and not take too much for granted when we hear of a patient having breathlessness, pain, or faintness.

This question of the proper analysis of symptoms of heart failure will be dealt with subsequently. They will here simply be enumerated.

DYSPNŒA.—In the first place, we must rely very largely upon breathlessness in our recognition of cardiac failure in later life, and we must be careful to put due weight upon this symptom and not to pass it by as not very important, because it is unaccompanied by any clearly marked signs of cardiac failure (*vide* p. 124). It may positively be the only well-marked symptom of an amount of fatty degeneration of the heart muscle that is seriously endangering the patient's life.

In any case where any dyspnœa exists in later life it is advisable personally to test the patient's power of walking upstairs or of fast walking across a room, and to note the amount of cardiac disturbance that results and the time that it takes for the heart to recover from the effects of the exercise.

It is hardly necessary in this connection to draw attention to the importance of making as sure as possible that the dyspnœa is of cardiac and not pulmonary origin; and if of cardiac origin that it is not due simply to reflex cardiac disturbance and therefore does not imply organic disease of the heart. (For the further discussion of this symptom, see p. 113.)

FAINTNESS.—In cases where the left ventricle is mainly affected, faintness, with or without actual fainting, is a very important sign of muscular weakness; but here, again, there is great liability of mistaking fainting attacks due to Menière's disease for those of cardiac origin, or, on the other hand, of mistaking slight attacks of faintness or vertigo, really due to failing heart, for reflex attacks of gastric origin. Such cases are described at p. 121.

WEARINESS AND LEG PAINS.—Associated with faintness there may be undue weariness on exertion and sometimes definite pains in the calves of the legs, which become so severe that the patient has to stop and rest for a minute or two before he can recommence exertion.

This is really one of the types of 'intermittent lameness,' and is due to the heart muscle being unable to keep up sufficient circulation in the muscles to supply their needs. Consequently, after walking a certain distance, fatigue is established and the exercise cannot be recommenced till the nutrition of the muscles has recovered itself.

For this reason, a man with myocardial weakness may be compelled to rest every two hundred or three hundred yards.

Such a patient, when asked as to his powers of walking, will tell you that when walking uphill he is stopped by breathlessness and has to rest, whereas on the level he has to rest 'because of tiredness,' and it is only on questioning him that he thinks it worth while telling you that the tiredness is really an uncomfortable aching in the calves of the legs. This subject is fully dealt with in the Essay on Leg Pains of Circulatory Origin.

In connection with these two symptoms of dyspnœa and faintness, it must be remembered that they give some indication by their relative amount as to whether the right or the left ventricle is the weakest.

A man with a strong left ventricle and a weak right one will be able to walk about and work till stopped by breathlessness, as in the case of the man referred to at p. 156; whereas, when the right ventricle is strong and the left weak the patient will only get breathlessness on very considerable exertion, and then of an alarming type, his ordinary activity being curtailed by weariness, faintness, or giddiness. This latter patient is far more apt to have the serious condition of his heart overlooked than is the former, and he is also more likely to die suddenly in an attack of fainting. Such a case is described at p. 124.

PALPITATION AND OTHER ABNORMALITIES OF RHYTHM.—Another symptom of importance is rapidity of the heart's action, often combined with irregularities of rhythm.

This symptom is of less value than the others given in the class of case we are dealing with, and great care must be taken to exclude the many causes other than myocardial weakness to which this symptom may be due.

The question of heart failure owing to irregularities of rhythm, heart block, &c., is too large a one to be touched upon here, although many of the cases may show early symptoms of a distinctive type.

The symptom of cyanosis may be of value, though it occurs as a late rather than an early symptom.

NO DROPSY.—Dropsy is a symptom which, in such cases as we are now speaking of, is always absent. The heart has not strength enough to keep up the venous plethora upon which it depends.

Its presence may help to distinguish the cases in the last group—namely, cases of strong heart without enlargement—from the cases of weak heart of which we are now speaking.

PHYSICAL SIGNS of Heart Failure in Weak Hearts.—The main signs upon which the diagnosis of myocardial weakness is based are, in later as in earlier life, those which show that less than the normal amount of blood is being kept in active circulation by the heart. And of these signs the ones which are due to emptiness of the great veins and of the blood-vessels of the thorax generally are by far the most important.

The limiting effect which rigidity of the fibrous tissues has upon the compensatory rise in the average level of the diaphragm has already been referred to. This interference with its compensatory elevation is especially noticeable on the right side, where the presence of the liver further interferes with its movement. So much is this the case that in the latter half of life it is not at all common to detect any elevation of the right half of the diaphragm as a result of cardiac failure.

On the left side the diaphragm is more free to move, and the level of the gastric resonance, as the patient lies on his back, is a very important guide to the amount of blood that is being kept in circulation in the thorax.

The occurrence of full gastric resonance in the fifth interspace is extremely suggestive of cardiac weakness.

Of almost equal importance with the change in the gastric resonance is the degree of hollowing or fullness of the supraclavicular spaces.

It is probable that in later life these spaces form a more useful guide than in earlier life; for in early life the ready yielding of the diaphragm, owing to the softness and elasticity of its fibrous tissues, will lessen the tendency to in-drawing of the tissues above the clavicle, whereas in later life the greater rigidity of its fibrous tissues will tend to accentuate that hollowing by limiting the compensatory movements of the floor of the thorax.

Certain it is that the careful and systematic study of the conditions observable at the root of the neck do give very valuable information as to slight changes in the heart's activity (*vide* p. 184).

It is also of importance to note the condition of the veins above the level of the clavicle, although in the cases now dealt with they are usually not distended. Emptiness of the neck veins is characteristic of great myocardial weakness. A further discussion of the venous phenomena of cardiac failure will be found at p. 182.

The size of the absolute dullness of the liver, as the patient lies in bed, also gives very valuable information as to the degree of fullness which the heart is able to maintain in the blood-vessels.

For even in later life—apart from such conditions as cirrhosis—the liver commonly seems to retain sufficient elasticity to show changes in the size of its area of absolute dullness when less distended with blood than normal; and in a case of myocardial weakness the variations in the position of the lower border of its absolute dullness are found to coincide with the variations in the strength of the heart.

The stronger the heart the lower the level, and the weaker the heart the higher will it be.

Quite late in life, however, one would not expect to see the extreme degrees of disappearance of the absolute dullness which are seen earlier in life.

Also on the left side the disappearance of the dullness of the left lobe of the liver is a very useful sign of heart failure.

As regards the pulse there is not much to be said; for, although when weak and small it gives very valuable evidence, when, on the other hand, it appears to be good it is a very uncertain guide; for, unless the usual character of the pulse is known, it is very difficult to say on first feeling a pulse whether there is anything about it to indicate heart failure.

The interpretation of the pulse in later life is very difficult and complex, and it is further discussed at p. 143.

And now as to the physical signs of severe heart failure that may be obtained from the examination of the heart itself.

When the heart is not very rigid, we may find—just as is the case earlier in life :—

Diminution in the area of cardiac dullness, and especially a raising of its lower border from rise of the diaphragm ; a weak or absent cardiac impulse is also of value.

Auscultation.—With regard to the heart sounds, the following points are of importance. In addition to their being simply weak, they are often also short, and do not give the impression of being produced by a properly vigorous contraction of the heart. Another important fact may be noticeable—namely, that in spite of the dyspnœa and the certainly existing embarrassment of the pulmonary circulation, there is little or no increase in the loudness of the pulmonary second sound or of the first sound audible over the right ventricle.

The absence of right ventricular pulsation and also of accentuation of the right ventricular sounds, in spite of the presence of distinct cardiac dyspnœa, may be taken as almost certain evidence of muscular weakness of the right ventricle.

When, in addition to this, it is found that the absolute cardiac dullness does not reach as far to the right as the edge of the sternum, but that resonance (due to the right lung overlapping the heart) is found at the sternal ends of the fourth and fifth left interspaces, the evidences in favour of a small weak right ventricle are very strong indeed.

If the importance of the diagnostic points here referred to was more generally recognised and more often looked for in doubtful cases of heart weakness in later life, the result would be that fewer cases of dangerous myocardial weakness would be missed, and fewer people would die sudden deaths from heart failure for want of warning as to what was safe for them to do and what was not.

In the more rigid type of heart most of these evidences of failure may not be observable, except the absence of cardiac impulse and the weak heart sounds ; and, if the patient is fairly fat, we may not be able to lay much stress on these.

Nevertheless, as a rule in later life, we are able to be fairly certain of the presence of myocardial weakness when we find heart failure associated with the following :—

Absence of venous plethora, as shown by empty neck veins and hollowing above the clavicle.

Weak or impalpable jugular bulb pulsation.

Gastric resonance higher than normal, especially if well into the fifth left interspace.

Smallness of the absolute liver dullness especially if it do not reach as low as the costal arch, and if the dullness of the left lobe be nearly or quite absent.

Diminution in the size of the area of cardiac dullness, with absence of the cardiac impulse and heart sounds weaker than normal.

In illustration of these points, the following clinical history is given, and the diagrams of the chest show clearly from day to day how these signs varied in a manner distinctly suggestive of their reliability.

HEART FAILURE WITHOUT ENLARGEMENT COMPLICATING PNEUMONIA

The following case illustrates well the importance of recognising the signs of myocardial weakness of which we have just been speaking :—

The patient was a man about forty, who was suffering from pneumonia of the right lower lobe, with some signs that the left lower lobe was also about to become involved. He had been only two days ill, and in spite of the severity of the local changes the temperature was only about 101° F., and the pulse about 100, occasionally rising to 110. The respirations, on the other hand, were fifty per minute, though this might partly be accounted for by the presence of a good deal of pleuritic pain.

There was very little cyanosis, although several times during the last twenty-four hours the patient had been decidedly blue.

The examination of the heart showed a condition that was not at all typical of so severe a case of pneumonia.

There was no cardiac dilatation, and the cardiac impulse was almost imperceptible.

There was no absolute cardiac dullness, and only a little relative dullness in the third interspace, and about two inches in the fourth. In the fifth interspace there was full gastric resonance up to the lower border of the fifth rib.

There was no absolute liver dullness in the middle line, but the full gastric resonance did not reach higher than about the tip of the xiphoid cartilage. The absolute dullness of the right lobe of the liver was also small and barely reached down to the costal arch and did not come to within an inch or more of the middle line.

The veins of the neck were not at all distended, nor was the hollow above the clavicles filled up. There was slight visible pulsation in the neck veins, but it was too feeble to be palpable, and the pulsation of the jugular bulb was also too feeble to be readily distinguished by the finger.

All the cardiac sounds were weaker than normal, and those over the right ventricle were slightly better marked than those over the left (see fig. 7).

Here, then, was a case where a very considerable degree of interference with the pulmonary circulation was unaccompanied by any distinct signs of venous plethora or of overaction of the right ventricle. Moreover, in spite of nearly the whole of the lower lobe of the right lung being functionally inactive there was not any increase in the size or activity of the left lung and there was, on the other hand, an actual diminution in the cubic content of the left side of the chest, as proved by the fact that the diaphragm was 1 inch or 1½ inch higher than normal on that side.

The marked rise in the level of the gastric resonance proved this to be the case.

Had the level of the gastric resonance and the size of the liver dullness not been looked for, the severity of the case might very easily have been overlooked. One might easily have given a very good prognosis, saying :—

'It is quite evident that the amount of damage to the lung is much less than the physical signs would lead us to believe, for the heart is not at all affected by it ; there is no fullness of the veins, and there are no signs that any extra strain is being thrown on the right ventricle—as evidenced by the absence of pulsation and there being no accentuation of the pulmonary and tricuspid sounds. The heart sounds at the apex are certainly rather weak, and the area of cardiac dullness not quite as extensive as normal—but this is easily explained by a compensatory enlargement of the left lung.'

In the light of what has been already said, the incorrectness of such an interpretation of the cardiac condition is manifest.

The case is really one of severe and dangerous myocardial weakness, and the further history of the case helps to explain the cause of the condition. Three weeks prior to the present attack, he had an influenzal attack accompanied by severe pains in back and limbs which kept him in the house ten days, seven of which were spent in bed. During the next ten days, he did his work in spite of great weariness and exhaustion.

This weariness probably meant that some myocardial weakness resulted from his influenza-like attack, and the toxins of his pneumonia attack had still further poisoned the heart muscle, thus bringing about the smallness and feebleness of the heart and the rise of the diaphragm which accompanied it.

The importance of the recognition of this fact is great ; for the danger of over-stimulating such a heart with digitalis would be very considerable. The immediate and the distant prognosis, too, are very different from those of a simple case of pneumonia. The risk to life is greater ; and if this be surmounted, the cardiac weakness would in all probability prevent his returning to work for months rather than weeks after his recovery from the pneumonia.

What has just been said about this case was written on the evening after the patient was first seen, and its correctness was proved by the further progress of the case which is here given, and the extreme weakness of the heart was shown by the fact that on the seventh day of his illness, when apparently going on quite well, a fairly severe attack of coughing nearly caused his death, for he became extremely cyanosed and pulseless, and appeared to be dying. He responded, however,

A CASE OF SEVERE PNEUMONIA WITH MYOCARDIAL WEAKNESS, WHERE VARIATIONS IN THE CARDIAC STRENGTH WERE SHOWN BY DAILY VARIATIONS IN THE SIZE OF THE CARDIAC AND LIVER DULLNESS AND IN THE LEVEL OF THE DIAPHRAGM

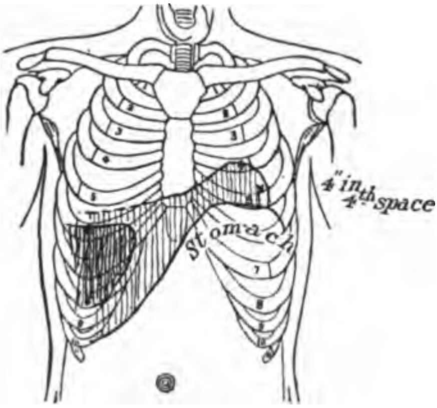


FIG. 7.

William H., aged forty, March 15, 1913. Extensive pneumonia of right lung. Note the high level of the gastric resonance showing diminution of the healthy left lung instead of the usual compensatory enlargement. Note also the smallness of the heart instead of the dilatation usual in severe pneumonia. These and other signs of lessened volume of the blood in circulation show the presence of dangerous myocardial weakness.

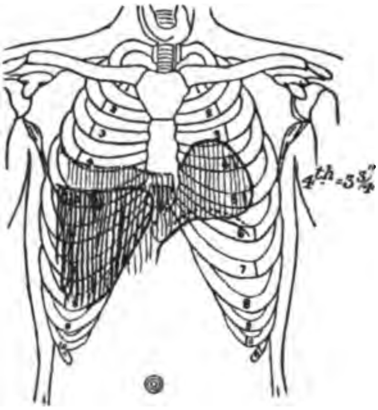


FIG. 8.

William H., March 19 (i.e. four days later). Cardiac and liver dullness increased, diaphragm less high on the left side, and other signs of increasing heart strength.

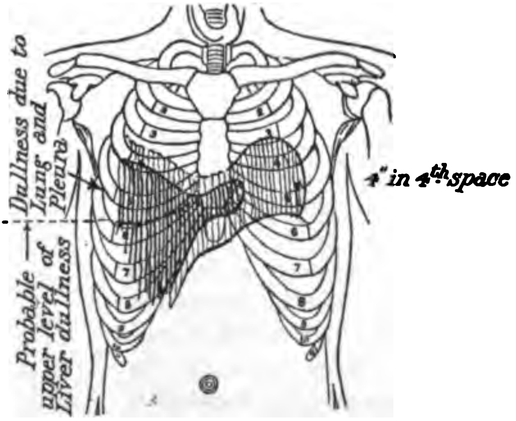

Dullness due to Lung and Pleura
Probable upper level of Liver dullness
4" in 4th space

FIG. 9.

William H., March 20. Increase of liver dullness. Onset of right pleural effusion.

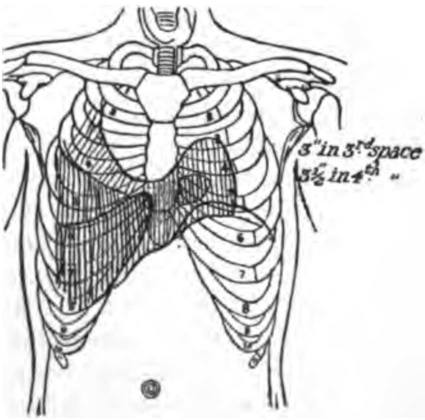


FIG. 10.

William H., March 21. Less cardiac dilatation. More pleuritic effusion and more consolidation of right lung.

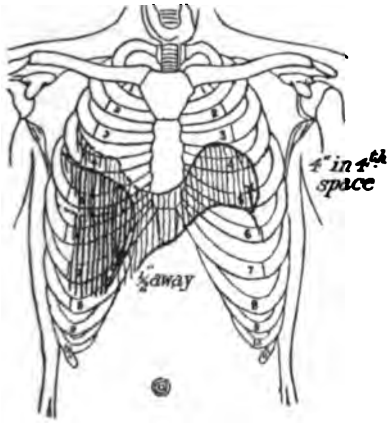


FIG. 11.

William H., March 22. Commencing absorption of pleuritic effusion and lessening of liver dullness from a rise of the diaphragm.

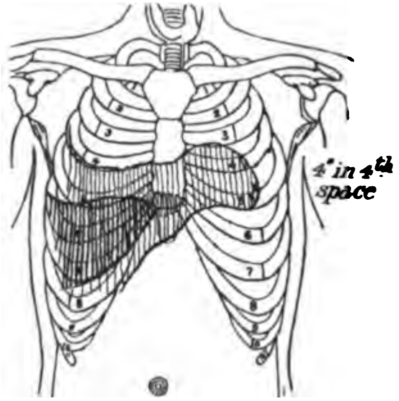


FIG. 12.

William H., March 24. Further absorption of the pleuritic effusion. Lessened liver dullness, probably from rise of right diaphragm. Sudden rise of blood pressure from 110 to 130, due to absorption of toxins from pleura, lead to some enlargement of the right ventricle.

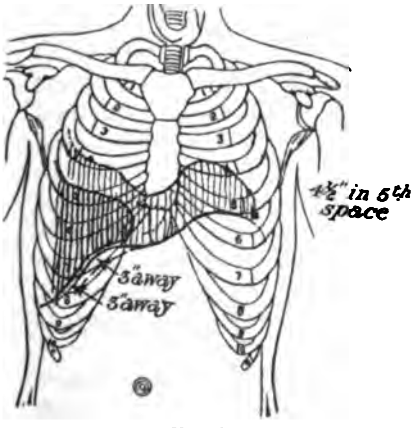


FIG. 13.

William H., March 25. Diminished cardiac and liver dullness. Blood pressure down to 126 mm. of mercury. Left ventricle a little larger. Right ventricle smaller again.

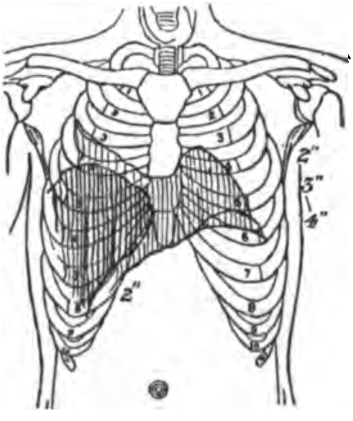


FIG. 14.

William H., March 27. Heart rather stronger and larger. Increase of dullness on the right is due to a return of the pleural effusion.

to free stimulation and to ether subcutaneously, and when the pulse returned it was found to be 148 per minute.

The case is so instructive that a series of diagrams of the changes in the physical signs from day to day, with short explanatory notes, are here given.

The further progress of this case is of great interest also in connection with the diagnostic point of which we are now speaking.

Faintness and weakness were the main features of his illness; he had not the restlessness and distress of an ordinary pneumonia.

During the next four or five days, he distinctly improved; the apex beat reappeared, and in three days had become well localised and fairly strong; the liver dullness had returned and was just at or below the costal arch, and the full gastric resonance was at the upper border of the sixth rib, although there was still some resonance to be detected in the fifth interspace (see figs. 8, 9, and 10).

Another interesting point was the increase of relative dullness in the third left interspace, showing that the heart was now strong enough to cause some dilatation upwards of its anterior wall. The apex at the same time was less far out.

Together with this manifest strengthening of the heart there was an increase in the area of the pneumonia. It first involved the middle lobe on the right side and then the lower and outer part of the upper lobe.

Next day, the eleventh day of his illness, he was not so well. The condition of the heart and liver suggested a loss instead of a gain in cardiac strength, for the absolute liver dullness was now half an inch above the costal arch, and the absolute cardiac dullness had gone; the blood pressure, moreover, had dropped from 118 mm. to 110 mm. (see fig. 11).

The condition of the lung posteriorly suggested a pleural effusion, and a puncture showed clear serum with flakes of lymph and abundance of pneumococci.

Two days later, there was a marked change in the physical signs, although the patient was slightly better on the whole.

The liver dullness, while good in the region of the xiphoid cartilage, was much less marked below the seventh rib; and next day there was full intestinal resonance below the seventh rib internal to the nipple line, and the anterior margin of the area of absolute dullness was three inches away from the costal arch and parallel to it; the cardiac dullness, too, was less and the relative dullness not reaching above the fourth rib. On the other hand, the heart was much stronger, the apical first sound being if anything louder than normal, and the right ventricular sound also good though not so loud. The pulse, too, was stronger, and the blood pressure 138 mm. (see fig. 12).

The explanation of these changes was given by examination of the lung; for not only had the dullness diminished anteriorly, as shown in the diagram, but on examining the back it was found that, instead of the dullness of the right side reaching to the middle line, or even crossing it towards the base of the lung, the whole of the spine was now resonant up to the level of the angle of the scapular; while above this level there was a band of resonance one inch wide to right of the spine, over which breath sounds, similar to those of the left lung, were audible.

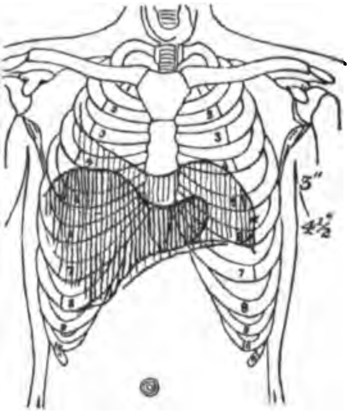


FIG. 15.

William H., March 29. Heart stronger and larger. Left ventricle dilated now, but pleural dullness increased.

There was no doubt that a considerable amount of serum had been rapidly absorbed from the pleura and, owing to the consolidation of the lung preventing its expansion, compensatory enlargement of the other lung had taken place and the diaphragm also had risen, thus lessening the amount of liver tissue left in contact with the lower ribs. The fact that the heart had not shifted towards the right, as well as the greater degree of crossing of the left lung posteriorly being above, instead of below, the level of the angle of the scapular, was explicable on the ground of the rigidity and possible adhesions of the affected right lower and middle lobes.

This theory of the absorption of the pleural fluid with its contained pneumococcal toxins was also suggested by the subsequent rise in the patient's temperature, his hot, dry skin, and also the sudden rise in the blood pressure.

My experience during the last twelve months has seemed to show that unrecognised pneumococcal infection—such as in frontal sinus empyema—is sometimes the cause of high blood pressure, and ordinary pneumococcal infection seems to have a special effect upon the pulse. The movement of the blood pressure in this case was certainly very striking—namely, steady at 118 mm. of mercury from the seventeenth till the twenty-first of March; 110 on the twenty-second; then on the twenty-fourth, signs that the serum had been absorbed with a blood pressure of 138; very vigorous cardiac pulsation and sounds, with strong venous pulse and fuller veins.

Next day, blood pressure 126 mm., followed in twenty-four hours by one of 120 mm.; but on this day evidence for the first time that the left ventricle was beginning to enlarge a little, the apex being 4½ inches from the sternum and palpable in the fifth interspace, when the ribs were elevated during inspiration, and in the fourth during expiration (see figs. 13, 14, and 15).

Subsequently, a small localised empyema formed at the right base, and was opened, and then a part of the consolidated lung broke down and suppurated instead of resolving.

The case illustrates the value of careful percussion of the heart and liver in estimating changes in the heart and chest.

The foregoing is but an illustration. Any case of heart failure due to influenza or rheumatism will show just as concise a picture as this or as that given at p. 196, and the certainty gained as to the condition of the heart well repays the few minutes necessary for the exact and careful mapping out by percussion of the area of absolute and relative dullness in the case of both the heart and liver.

This case also shows a trace (the man was forty-two years of age) of the dilatation of the anterior wall of the right ventricle, which is spoken of as one of the most certain signs of the return of strength after a myocardial affection where the heart is sufficiently distensible.

The disappearance of the lower half of the liver dullness, owing to the rise of the diaphragm, is also a good illustration of this phenomenon.

The following case also illustrates the fact that myocardial weakness, with or without the relative cardiac rigidity of later life, gives a different picture in the case of pneumonia from that afforded by a patient with a strong heart in early life.

In this latter case, we can rely upon the loudness of the right ventricular sounds, the amount of cardiac pulsation over the right ventricle, together with the degree of fullness of the veins and the amount of pulsation in them, as indicating in a reliable manner the degree in which the pneumonia is interfering with the circulation and as showing the severity of the case.

Thus, supposing we are dealing with an influenzal or a catarrhal case, where there is some tubular breathing over the lower lobe of the right lung with weak and wheezy breathing and some slight impairment of the percussion note at the left base, combined with a quietly acting heart showing neither dilatation of the right ventricle nor any evidence of its failure, by fullness or pulsation of the neck veins, we should be inclined to say that the pneumonia was not of a very serious type, and that the prognosis was good. If we were dealing with a young and fairly strong patient, this diagnosis would probably be correct. If, on the other hand, the patient was well past middle life, we might be making a dangerous mistake by reading the symptoms thus.

Here is such a case. An elderly lady suddenly developed a severe feverish cold with bronchitic symptoms. On the second day signs of influenzal pulmonary catarrh were present. On the fourth day the following condition was present. There was some consolidation with nearly tubular breathing along the upper border of the right lower lobe posteriorly. Over the greater part of the left lower lobe, posteriorly, the breath sounds were weak and wheezy and the resonance a little impaired. As to the heart, there was no increase in the loudness of the right ventricular sounds, and there was no pulsation over the right ventricle, visible or palpable, and no signs of dilatation, and the condition of the right ventricle was much the same as it was before the attack. There was no increased fullness of the neck veins, and little, if any, pulsation—just as was the case prior to the attack. The pulse was, however, bad, and the left ventricle more dilated than was usual with the patient. Now, how were these signs to be interpreted? Did this quietly acting right ventricle imply that the pneumonia was only slightly interfering with the circulation through the lungs and that the right ventricle was in a satisfactory condition? By no means. Had I not known what the usual loudness of the right ventricular sounds were, I might possibly have been deceived and taken this view; but as it was, the recognition of the fact that the right ventricular sounds were rather *less* loud than was usual in this patient, showed what was really taking place —namely, that the pulmonary circulation was so embarrassed that the somewhat feeble right ventricle could not cope with it, and there was a great risk of fatal dyspnœa. This was shown also by an alarming attack of breathlessness which followed upon some slight undue exertion early on in the illness. It was also proved by the fact that when the lung began to clear, the right ventricle became stronger again, and its sounds were louder and pulsation became evident just to the left of the sternum. The mode of resolution proved as was suspected—that the affection of the left lung was really a central consolidation with blocking of the bronchi; for, as resolution proceeded, a certain amount of faint tubular breathing became audible, and the whispered voice sound was far too audible over much of the lower lobe (*vide* p. 140).

In such a case as this a misreading of the heart condition early on might have easily led to a fatal result, through allowing the patient to attempt exertion—such as getting out of bed.

Difficulties in diagnosis may arise in cases where, in later life, myocardial weakness from muscular degeneration or from the toxins of infective disease occurs in a heart already enlarged from valvular disease and overstrain. But here the general principle of looking for signs of lessened vigour, and that the heart is not able to keep in circulation the amount of blood that it has been in the habit of doing, is just as reliable as it was in the cases already described, where there was an onset of muscular failure in a heart that was previously normal.

A lessening in the loudness of the murmurs, a diminution in the vigour of the venous pulsation in the neck and of the size of the enlarged liver, would all point to the onset of myocardial weakness.

In order to illustrate further the difference between cases of heart failure with enlargement, and heart failure without enlargement, the two following clinical pictures are given:—

The two patients here referred to were both seen on the same morning and in consultation with the same doctor.

Both were ladies over eighty, and both were hindered by breathlessness and weakness from taking much exercise. The elder of the two, a lady of eighty-five, was vigorous in mind and body for her age, and able to go upstairs and do a certain amount of walking on the level, but any exceeding of the narrow limits which were set her was sure to produce exhaustion and some dyspnœa.

In this case the heart showed what may be called the ordinary signs of heart failure with enlargement. The apex was in the sixth space, 5½ inches from the middle line, i.e. close to the anterior axillary line. Its impulse was well defined, and fairly well localised. The right ventricle, too, was strong, and its impulse would show clearly in the fourth and fifth interspaces, near the sternum, on any overtasking of the patient's strength. There was a well-marked but not very loud mitral systolic murmur, and a loud superior vena cava murmur heard to the right of the sternum up to the aortic area.

The neck veins were not distended, but pulsation was fairly well marked in the jugular bulb.

The pulse was full and strong—too full between the beats—and suggestive of arterio-sclerosis, were it not that the wall was soft and of good elasticity. The heart rhythm was at times slightly irregular, owing to occasional extra systoles following close on the preceding first sound, and almost certainly what may be called aspiration extra systoles, and due to mechanical stimulation of the left ventricle by the inrush of aspirated blood, and implying in this case dilatation of the left auricle (see p. 497).

The blood pressure was 170 mm., and the pulsation of the mercury column markedly lessened below 120 mm., suggesting that this was the diastolic pressure.

This is an ordinary case of heart failure with dilatation secondary to high pulse tension, and in this patient the occurrence of exhaustion rather than dyspnœa showed that the left ventricle was more faulty than the right; the same fact was pointed to by the occurrence, when the patient over-exerted herself, of signs of increased right ventricular action —namely, the appearance of a right ventricular impulse and an increase in the loudness of the right ventricular sounds.

In contrast with this was the second case seen. Here the lady was also over eighty, and was also active in mind and body, although for some years past she had been hindered by marked dyspnœa from any continuous or severe exertion. One-half or three-quarters of a mile on the level was all the exercise she could take without the occurrence of troublesome dyspnœa.

Her pulse was in marked contrast to the last patient's. It was very small, but regular, and there was no marked diastolic over-fullness of the vessel as in the other case, enabling the artery to be rolled under the finger. The blood pressure was 152 mm. of mercury, and the diastolic pressure was 100. There was no pulsation visible in the neck veins, and there was distinct hollowing above the clavicles, suggesting emptiness of the veins. This was confirmed by palpation of the jugular bulb, for its pulsation was not well marked.

On examining the heart, no pulsation could be detected anywhere, and the apex beat could not be felt. The cardiac dullness, too, was decidedly small, and the diaphragm high, as judged by the level of the gastric resonance, which reached to the fifth rib in the left nipple line. The liver dullness, too, was much smaller than normal, and well-marked intestinal resonance was obtainable, as the patient lay in bed, for more than one inch above the right costal arch. In the other patient the liver was very nearly normal in size and the gastric resonance was normal.

The heart sounds were weak, and there was no murmur present.

An interesting feature about the case was that in spite of the quietness of the heart's action and sounds, and the smallness of the pulse, the pulsation of the carotid arteries was strong and vigorous, much more so than normal, and the artery was full and fairly hard throughout the diastole, as in cases of arterio-sclerosis.

The condition of the carotids, as compared with the pulse, suggested the probability that the smallness of the radial artery was a compensatory change secondary to the cardiac weakness, and that with any increase in the heart's strength there would be a marked increase in the volume of the pulse. In answer to this suggestion, the practitioner in charge of the case said that the improvement in the volume of the pulse *was* sometimes remarkable, and that sometimes the pulse was very much larger than it was at the time of examination.

As regards this case, it was quite evident that of the two sides of the heart the right ventricle was the weaker, and the condition of the circulation pointed to a somewhat considerable amount of degeneration of the muscle of that chamber. The next question was whether this was the only serious cause of interference with the heart's action.

The full and sustained carotid pulse suggested the possibility that there was also some rigidity of the first part of the aorta, because were the first part of the aorta normal there was no reason in such a case as this for the larger arteries showing more pulsation than normal, while the smaller ones showed less. In spasmodic contraction of the peripheral vessels such a condition might be possible, but there was no suggestion of this here, as there was no sign of any faulty circulation in hands or feet. The colour and warmth of hands and feet were always good and the dilatability of the radial artery when the heart was excited negatived the presence of muscular spasm.

The absence of any dilatation and hypertrophy of the left ventricle—such as usually accompanies a loss of elasticity of the first part of the aorta—would in this case be prevented by the faulty nutrition of the cardiac muscle, and therefore weakness and breathlessness would advance *pari passu* with the change in the aorta and not be warded off for a time by muscular hypertrophy.

Some other explanation might, however, be found for this phenomenon.

Of these two cases, the first was one of simple overstrain from high arterial tension in a heart whose muscular tissue was fairly sound considering the patient's age, and was able to develop an intraventricular pressure in excess of the resisting power of the weaker portions of the ventricle walls, thus causing dilatation. In this case there was no great risk of sudden death.

In the second case, the primary cause of the cardiac failure was muscular degeneration involving the right side of the heart to a much greater extent than the left.

Whether previously there had been any cause for embarrassment—such as high arterial tension or rigidity of the aorta—it was not easy to say, as the myocardial weakness would prevent the characteristic signs from showing themselves. The smallness of the pulse, and the emptiness of the veins and other signs of a diminution in the volume of blood in active circulation, were a measure of the muscular feebleness of the heart, and in this connection it was an interesting fact that the patient was an extremely small eater, her daily output of urea being 150 to 200 grains only—as if nature were calling for a reduction in the intake of proteids in order to assist in keeping the volume of the blood below normal.

The other patient had a good appetite, and her intake of flesh food had to be compulsorily lowered in order to lessen her purin intake and keep down the blood pressure.

EARLY DIAGNOSIS OF HEART FAILURE IN CASES OF VALVULAR DISEASE IN LATER LIFE

The same principles are applicable to the recognition of muscular failure in a heart that had previously been enlarged as a result of valvular disease or overstrain, as those here laid down for the recognition of cardiac failure in a previously healthy heart. In a heart dilated and hypertrophied from either of those causes, the onset of muscular failure would be accompanied by a lessening in the vigour and amount of the venous pulsation in the neck and by a lessening of the loudness of the heart sounds. There might also be a lessening in the amount of dilatation of the right ventricle, and this change, together with the lessened pulsation in the neck, might very readily be misinterpreted and taken to be a good sign instead of a very bad one.

A case is mentioned on p. 63, where a sudden diminution in the size of a distended liver was a sign of sudden heart failure from over-stimulation of a weak and failing heart. Such diminution in the size of a liver enlarged by tricuspid regurgitation shows a lessening in the amount of tricuspid leakage, and might well be due to lessened dilatation and recovery of the ventricle; but it must clearly be borne in mind that lessened tricuspid regurgitation may also be due to the onset of muscular weakness, and a sign of failure, and not of a gain in its muscular strength.

The changes described in the case of penumonia on p. 97 illustrate very well what might be expected to occur in a case of an overacting right ventricle when, after a time, muscular feebleness began to develop.

As the general principles both of heart failure in early, in adult, and in later life are fully applicable to these cases where muscular weakness appears in a heart previously dilated or hypertrophied as a result of valvular disease or overstrain the subject need not be dealt with in greater detail. It is hardly necessary to point out the importance of early recognising muscular failure in cases of valvular disease. Our treatment of such cases often has to include powerful heart tonics, in order, for instance, to raise a falling blood pressure up to the level necessary for cardiac nutrition, or in other cases so to raise the intraventricular pressure that it may speedily bring about that dilatation upon which compensation depends. In all such cases, we must watch very closely lest we go too far and stimulate the heart to an expenditure of energy which is in excess of what its powers of nutrition can make good. The careful watching in such cases for the first signs that the muscles of the heart are becoming weaker from over-stimulation is therefore of the greatest importance. The early detection in valvular disease of signs of muscular weakening, which disease of the coronary arteries slowly induces, is also of great importance; for a heart whose nutrition is not good will naturally not stand stimulation as well as one whose nutrition is sound.

Another case where information of great diagnostic value was gained from the size of the liver and heart is the following:—

A. H., aged forty-four, commercial traveller, who used to do 20,000 miles a year in spite of suffering from aortic regurgitation of a moderate degree. When I first saw him, he was suffering from failing compensation with a dilated right ventricle, breathlessness, and pain, but no dropsy. I saw him again six years afterwards and found him to be suffering from palpitation and breathlessness and total inability to continue his work—probably the result of influenza. On this occasion, although his cardiac symptoms were quite as severe as when seen six years earlier, the cardiac apex was not as much outside the nipple line by one inch, and instead of there being marked dilatation of the right ventricle the area of cardiac dullness was quite small. Instead of the stomach level being nearly normal (at the level of the sixth rib) there were two inches of gastric resonance in the fifth space. The liver dullness also was small in this last attack—not reaching the costal arch by one inch. There was no fullness of the veins of the neck.

Here we are dealing with a case of aortic regurgitation with breathlessness, but without cardiac dilatation or distension of the veins. On the other hand, there was smallness of the heart, smallness of the lungs as well, as judged by the rise in the gastric resonance, although the upper level of the liver dullness was normal, and also smallness of the liver dullness.

It was quite evident that the man was suffering from pure myocardial weakness and not the ordinary mechanical failure with a strong cardiac muscle, which is the usual cause of heart failure in aortic regurgitation.

The prognosis for the future was therefore much worse, and the need for cutting down his work quite imperative. Doubtless, the same estimate of his condition might have been arrived at without noting the smallness of the liver and the rise of the gastric resonance; but these two indications of weakened circulation were of great value.

Short Notes upon some of the Diagnostic Difficulties associated with the Recognition of Heart Failure in Later Life.—The main cause of the difficulties which accompany the recognition of heart failure in later life is to be found in the fact that muscular feebleness plays a very important part in that failure, and we cannot expect that any striking positive signs—such as loud murmurs, dilated auricles or veins, or well-marked pulsations—will characterise feeble cardiac action.

The physical signs of muscular failure must therefore be, perforce, negative in their nature. Consequently, we have, as already pointed out, to rely much more upon symptoms than upon positive signs for the diagnosis of this type of heart failure in later life. Symptoms are, however, much more likely to be misleading than physical signs, owing to the simulation of symptoms due to organic disease by those due merely to reflex disturbances.

This section will therefore be commenced by a few notes upon the various symptoms, and with illustrations of some of the errors in diagnosis that have to be avoided.

But here a word of apology must be offered for fear anything approaching completeness be expected in the following pages. The subject dealt with is so complex and so difficult, and our knowledge in many respects so limited, that nothing approaching completeness or scientific method can be attempted by the writer. He can, at most, give some illustrations gathered from his own experience of some of the pitfalls into which the practitioner and the specialist may fall when face to face with cases of heart failure, or of supposed heart failure, in later life.

There is plenty of scope for a whole book being written upon this one subject alone.

DYSPNŒA

Firstly, as to dyspnœa—for this sign is our sheet-anchor in dealing with cardiac failure in later life.

It is hardly necessary to point out the necessity for making certain that the breathlessness is of true cardiac origin, and not due to damage to the lungs--an error that can easily be made by any of us from imperfect observation in a case when our attention is absorbed by some apparently obvious cardiac abnormality (*vide* the case described on p. 374). Upon another point we must convince ourselves—namely whether the dyspnœa, although certainly cardiac in origin, is or is not secondary to tachycardia, or, in common parlance, 'palpitation.' In other words, ascertain whether the breathlessness is a constant symptom, or whether at times it is much less troublesome than it is at others. In the latter case it may be simply secondary to a palpitation of reflex origin, as in the case described on p. 115.

Again, if the dyspnœa be a constant phenomenon it may yet be associated with a tachycardia—such as that symptomatic of Graves's disease, or due to some other cause, and not directly due to organic cardiac failure.

A case may, however, occasionally be met with where the tachycardia and its accompanying dyspnœa may both be associated with overstrain, and may represent organic damage to the heart. Such a case is given at p. 324.

Dyspnœa when a prominent symptom suggests myocardial failure of the right rather than of the left ventricle, see p. 154.

PALPITATION

Another important sign of heart weakness is palpitation, although it is very commonly in later life associated with causes other than organic disease—such for instance as Graves's disease and reflex disturbances. Moreover, in pure muscular failure the rapid conscious overaction of the heart, which patients call palpitation, is not a very characteristic phenomenon.

Often associated with palpitation are the many cases of cardiac irregularity, some of which are due to organic damage to the heart, as in the case of the various types of irregularity with which the name of Sir James Mackenzie will always be associated, and some of which are purely functional in origin.

Into the former group I shall not enter here, as the subject is too large a one, and is, moreover, being very fully dealt with by very many writers at the present time.

It may be of interest, however, in this connection to give some illustrative cases: in one of which, at all events, an error in diagnosis might well have been made, had the patient been a dozen years older.

Mr. W., aged about forty, a dental practitioner, came to me complaining of extreme cardiac discomfort, with a most trying sense of impending death. The heart was almost continuously irregular, and at times when walking or exerting himself, he was seized with violent palpitation and irregularity; he was conscious of individual beats of the heart so forcible that he felt as if they shook his whole chest, and these were preceded or followed by beats of extreme rapidity, or by a sensation that his heart was stopping altogether and that he was going to fall dead.

Careful examination of the heart, however, showed no trace of any abnormality in the size or sounds of either ventricle or in the venous or arterial circulation, except the arythmia already described. Moreover, on testing the strength of the heart by making the patient ascend stairs, there were no signs of any failure in its strength, so long as the palpitation was not extreme at the time. A good prognosis was therefore given.

This view of the case was borne out by the history, for, although the man paid me very frequent visits for some months in order that I might examine his heart and reassure him that he was not going to fall down dead in the street, after having had it for many months the palpitation suddenly left him: so suddenly, in fact, that he could point to the spot where he was when the sensation came over him that his palpitation had gone; and strangely enough it happened as he was walking along the street not two hundred yards from my consulting-rooms.

The following case also is one which presented a good deal more difficulty, and is of very considerable interest.

The patient was a lady about forty years of age, who for two years had been working far too hard in connection with a philanthropic and religious organisation in which she was interested. Her work involved physical as well as nervous strain; for she not only conducted frequent meetings—speaking to various types of audiences—but also often had long days and sometimes, owing to absence of other means of transport, had herself to carry a heavy bag to or from railway stations or meetings. When she consulted me, her chief trouble consisted in neurasthenic symptoms combined with most troublesome palpitation and breathlessness. The history seemed to point to heart weakness, in which nerve strain played an important part, as the cause of her symptoms.

This diagnosis appeared to be confirmed by the examination of the patient. She was thin. The heart was rapid and the pulse wave was poor and jerky in character. The veins of the neck were a little full, and there was a slight amount of true pulsation in them ; there was also some excess of arterial pulsation suggestive of the kind observable in Graves's disease. There was no dilatation of the heart, the apex being in the nipple line, and there was no abnormal cardiac pulsation. The cardiac impulse, though forcible, was not well sustained, and the first sound of the heart, though loud, was unduly short and jerky, giving an impression of ineffectiveness. The patient complained often of feeling her heart thumping in her chest, and she also had palpitation and breathlessness on exertion—such as running, or on hurrying upstairs. Palpitation and dyspnœa on exertion were the chief symptoms of which the patient complained, and it looked very much as if it were a case of muscular failure of the heart without definite enlargement, resulting from a combination of nervous and physical overstrain.

She was treated by rest and tonics under thoroughly congenial surroundings for some weeks, and belladonna was given in view of the palpitation and the other symptoms suggestive of Graves's disease. Nevertheless, after six weeks, she still complained that any slight exertion—such as lifting a child into a chair—would be apt to start an attack of palpitation and breathlessness that would last for some hours, or any sudden call upon the nervous system—such as seeing an unexpected friend or playing a somewhat difficult hymn-tune on the piano before the family she was staying with—would have the same effect.

There was, however, one symptom which was not quite in keeping with the above diagnosis, and it was not until the symptoms failed to yield to treatment that seemed eminently calculated to relieve them that its importance was recognised. This was the fact that the palpitation would sometimes come on at night or in the afternoon or evening when no cause at all for it was apparent. Other facts also were elicited, which if the patient had remembered earlier on would have helped to clear up the diagnosis. For instance, she stated that riding in a shaky motor-bus always made her feel bad, and that once it so upset her heart that she nearly fainted and had to get out. This is a most characteristic symptom of irritability of the transverse colon, the vibration setting up in it spasmodic contractions which give rise either to pain or simply, as in this case, to reflex disturbances (see also case at p. 130).

She also stated that the thumping of her heart was apt to be bad two hours after a meal.

In consideration of the fact that circulatory disturbance might have been of reflex origin, and due to abnormal irritability of the colon, the abdomen had several times been carefully examined, and although on one or two occasions a slight amount of colon irritability was observable, it was apparently insufficient to justify a diagnosis of reflex cardiac disturbance. About the time, however, that the history of nocturnal attacks of palpitation was obtained, the transverse and ascending colon was on one occasion found to be markedly hard and tender in places. The treatment of the case was therefore changed, and definitely cardiac remedies were replaced by fairly full doses of hyoscyamus with an intestinal antiseptic. This treatment had an almost immediate effect in the relief of the patient's symptoms, and she at one began to improve. After only a week or so she expressed the opinion that I had now found the right medicine for her.

While freely recognising the possibility of error in ascribing any particular result to any particular remedy, there was in this case abundant reason for coming to the conclusion that the cardiac symptoms, though pointing strongly to genuine cardiac failure, were in reality only of reflex origin and secondary to abnormal irritability of the colon. Another point in this case was the presence of well-marked colon irritability without the presence of an adequate amount of pain, thus suggesting that the afferent impulses, which ought to have given rise to pain and were not doing so, were producing nervous phenomena of some other description.

Palpitation as a cardiac habit persisting after the recovery from myocardial weakness.—When dealing with the subject of leg pains of circulatory origin a remarkable case is given where a disturbance of the circulation in the legs, lasting six months, was evidently due to the retention of abnormal action on the part of the vasomotor mechanism as a matter of habit.

The vasomotor mechanism had got into the habit of a sluggish and incomplete response to the calls for vaso-dilatation which came from the muscles during exertion, and the habit was cured by increasing the stimulus to vaso-dilatation by greater violence and suddenness of movement.

Clinical experience shows that the cardiac mechanism also may suffer in the same way, and that after a period of extreme weakness from myocardial disease, the palpitation and faintness which were symptomatic of the weakness, may be retained by the mechanism as a habit after the heart has regained its strength. In such a case as this, reliance has to be placed on the cardiac phenomena which show return of strength in the heart wall, and the symptom must be ignored from the point of view of diagnosis, in spite of the protestations of the patient.

Small Heart in Myocardial Weakness

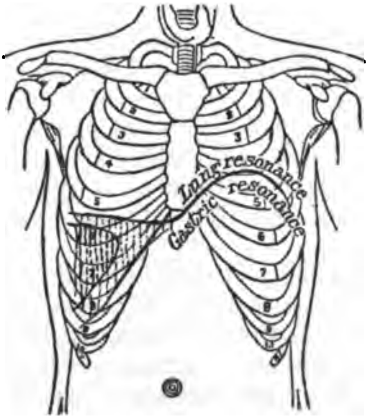


Fig. 16.

Dr. G., aged twenty-six, March 24. Myocardial weakness after diphtheria. No cardiac dullness at all. Gastric resonance up to the fourth rib. (Absolute liver dullness approximate only.)

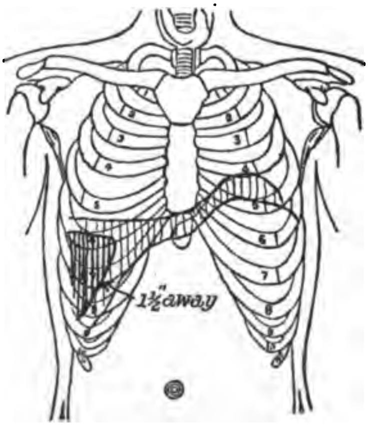


Fig. 17.

Dr. G., April 8. Heart now shows a trace of relative dullness in the fourth space and a little right ventricle pulsation. Still some gastric resonance up to the fourth rib.

To do so requires considerable boldness on the part of the physician, for it is not as a rule wise in a case of myocardial weakness to encourage and almost force the patient to get out of bed, in spite of the fact that it brings on troublesome palpitation or even a sense of faintness and fear of impending death.

It was only the absolute reliance of the writer upon the signs of heart weakness, which are described in the preceding pages, that gave him confidence in ignoring the symptoms of heart weakness in the two cases which are here recorded, because they were in conflict with the physical signs of returning cardiac strength. The first case is the young man, whose case is shortly reported on p. 182 as an instance of high diaphragm and small heart in acute myocardial failure after influenza. When at its worst the heart weakness was so great that faintness and even definite fainting attacks were caused by the least exertion. The patient was even afraid to lift his arm as he lay in bed because of the faintness which the exertion produced. The heart, however, gradually recovered its strength, and in the course of some weeks the cardiac dullness had nearly returned to normal. He still had, however, the same feeling of faintness, and was afraid to make any effort.

Relying, however, upon the physical signs of returning strength, he was encouraged to make more effort; but so marked were his symptoms that he finally had to be compelled to exert himself, and it was only after finding that his symptoms were not increased by exertion that he was willing to accept his doctor's advice.

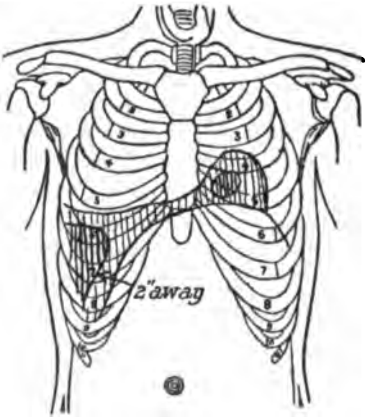


FIG. 18.

Dr. G., June 6. Heart stronger; cardiac dullness larger; gastric resonance lower; liver dullness still very small.

Another similar case is that of a lady (a doctor) who had a severe attack of myocardial weakness after diphtheria. The case has already been referred to at p. 68 as a good illustration of heart failure with small heart, and is worth recording in some detail here.

The patient was a lady aged about twenty-six, who had had an attack of diphtheria six weeks previously. She had no paralysis, but had had heart attacks since her illness. She was very weak, and any exertion would bring on attacks of cardiac irregularity and intermission, which made her afraid to move at all. She had been confined to bed since her attack of diphtheria. The pulse wave was of very poor volume and weak, but the artery was kept relatively full; a weak pulse of relatively high tension, such as is often found in myocardial weakness. On examining the heart, no relative cardiac dullness could be detected, and the gastric resonance was up to the fourth rib, giving the appearance shown in fig. 16. The heart sounds were very weak and wanting in tone, and there was no trace of cardiac pulsation. The liver was not carefully noted for fear of disturbing the patient. In the figure it is drawn the same as at the next visit. She was given calcium lactate. When seen a fortnight later, she was better. There was a trace of relative cardiac dullness and of pulsation in the fourth interspace, but the resonance of the stomach could still be detected up to the lower border of the fourth rib. The pulse was a little fuller and the heart sounds not quite so weak. I have no note of the veins of the neck, but I remember them as being empty with much hollowing behind the clavicles (see fig. 17).

The patient progressed slowly but steadily, and by the beginning of June she was decidedly stronger. The relative cardiac dullness now extended for some 2½ or 3 inches in the fourth interspace; the apex was just felt in the fifth space, and there was a trace of pulsation in the third interspace. The heart sounds were much stronger, as was also the pulse, though the blood pressure was now only 90 mm. of mercury. The liver had not increased in size, though the gastric resonance now only reached to the fifth rib (see fig. 18).

After two or three weeks more, the heart was evidently stronger, and by the examination of the chest I was so sure of this, that I advised the patient to get up, though she was very reluctant to do so, because such a little exertion give her cardiac discomfort. The first few attempts at getting up brought on attacks of palpitation and cardiac discomfort, and made her afraid to attempt to do so any more. An examination of the heart, however, showed that even when she felt ill it was not materially affected, and she was therefore advised to continue getting up, but was told that when the attacks came on she must just rest till they passed off. She was sent away to the seaside before she herself felt fit, but with no bad results, and at the end of July reported having had no heart attack for several weeks, but that her powers of walking about were still very limited, and she had to use a Bath chair a good deal. In this case the irritable weakness of the heart remained as a cardiac habit after the muscle had really recovered itself and if in this case the medical adviser had been guided by the patient's symptoms of cardiac distress her recovery would in all probability have been delayed many weeks and possibly even months.

FAINTNESS

The symptom of faintness, important as it is in the diagnosis of cardiac failure, is, nevertheless, one whose true value it is very difficult to appraise ; for there are other causes of recurrent fainting attacks, such as might be due to cardiac failure. Of these by far the most important is labyrinthine disturbance, and that associated with high blood pressure is specially likely to give rise to an error in diagnosis. The misleading points in such a case are the suddenness of the attack, which may come on without any warning, and consists in a simple faint without any vertigo ; it is, moreover, usually followed by a sense of extreme weakness and prostration, and the patients will often not believe that the weakness is of nervous and not of cardiac origin. The points in favour of the case really belonging to what is commonly called Menière's disease are these : (1) sudden rotation of the patient will in such a case be very likely to demonstrate the presence of vertigo, and thus help to clear up the case ; (2) the occurrence of vomiting or nausea with the fainting attack is very suggestive of its cerebral origin, although vomiting may accompany an ordinary cardiac fainting attack ; (3) the presence of vertigo after the attack is often a valuable aid to the formation of a correct diagnosis. In addition to these positive symptoms, the endeavour must be made to exclude true cardiac weakness, and in this connection the old proverb comes to our aid : 'The strength of a chain is that of its weakest link.' If the patient can ever walk upstairs easily without breathlessness, or walk up hills at a good pace, the faintness is not of organic cardiac origin, and there need be no fear as to the presence of fatty or other degeneration of the heart. Here is a striking case where an error of diagnosis deprived a man of many years of usefulness.

The patient was a gentleman aged sixty-nine who had suffered from severe attacks of faintness and giddiness for four five or years or more. They were not very frequent—sometimes once a week—and sometimes he would go for months without one.

He saw a consultant who had a well-earned reputation in connection with cardiac diagnosis and treatment, and was told that his attacks were mainly of cardiac origin, and I believe also there was some suggestion that disease of the vertebral arteries, by further interfering with the blood supply to the brain, had something to do with the severity of his fainting attacks.

He was told that any one of these attacks might prove fatal; was told to give up all his public work, in which he was much interested, and live the life of an invalid; and for three or four years he lived in the constant expectation of sudden death (I will not say fear of death, for he did not fear it). His wife, too, was prepared to have him brought back to the house dead every time he left it. A truly tragic and painful experience. Some five years or so after the original diagnosis was made, he came to me, and I was surprised to find that in many respects his attacks were typical of Menière's disease, and not of fainting of cardiac origin; moreover, on questioning the patient, I could find no evidence of there ever having been marked dyspnœa or exhaustion on ordinary exertion even when the attacks were at their worst, nor were they definitely brought on by exertion, so far as I could ascertain. There was, moreover, a considerable degree of deafness in the ear, and the Eustachian tube on that side was quite obstructed and no air could be got in with the Eustachian catheter. As I had the catheter in place, I passed a bougie into the tympanum, and this single opening up of the tube seemed to suffice to relieve him. At the time of his visit to me, he was only having attacks every few months, and after his visit to me I believe he did not have another attack.

I of course told him my view of his case, and he at once resumed the degree of activity which his age rendered advisable, and without any unsatisfactory results.

Here is another case somewhat similar. Mrs. D., aged sixty-one, a well-nourished woman of fairly healthy appearance and with no definite signs of cardiac failure, complained that two years ago she had a bad fainting attack on first getting out of bed in the morning. A month or two later, she had another, falling unconscious on the floor of her sitting-room as she was about to go out of the house. The attacks since then have occurred every few months—sometimes the interval will be one month, sometimes three; but until August 1912, when she had two in one week, the interval had never been less than a month. From January to March 1913, she only had three attacks. She was afraid to go out into the street for fear of one coming on; but so far this had not happened, the attacks mostly being in bed or early in the morning. This history of course pointed clearly to the attacks being either mild epilepsy or of labyrinthine origin.

That they were not cardiac was shown by the fact that exertion never caused them; that the patient, though rather breathless on walking uphill, did not suffer at all from serious dyspnœa, and could go upstairs fairly easily. She was convinced that her heart was at fault. 'Her circulation was bad.' The fingers were very apt to go dead in cold weather, and she at times felt weak and ill. The heart certainly was not strong, but it was not weak enough to justify a diagnosis of the fainting attacks being due to myocardial weakness. The cardiac area was only a little smaller than normal; the sounds only a little weak, but the jugular bulb pulsation was of fair strength. The gastric resonance was a little high—i.e., to the upper border of the sixth rib—and the absolute dullness of the liver a little diminished in size. The blood pressure was 170. The daily excretion of purin bases was 0·46 instead of being under 0·2 as it should be, and there were 580 grains of urea secreted in the twenty-four hours.

On testing the semicircular canals there was distinct hyperæsthesia to rotation in the positive direction, the after-sensation lasting much longer than the original rotation; while for negative rotation the after-sensation was about normal. Although she was only rotated slowly (three seconds or so being taken for a rotation through three-quarters of a circle), and the test was only once repeated, there was a little unsteadiness in the carriage of the head afterwards and the patient complained of feeling very 'giddy and horrid,' and afterwards she said she felt confused and hardly able to hold her head up.

In addition to the high blood pressure and abnormal intake of purins there was also a considerable amount of colon tenderness and irritability, and quite enough to be a possible cause of considerable reflex cardiac or circulatory disturbance.

A confident diagnosis was given in this case that the patient need not fear as to her heart. In a case like this it is probable that local treatment directed to the opening of the Eustachian tubes would be necessary as well as that directed to the lowering of the blood pressure by lessening the abundance of the waste products of metabolism in the circulation.

Another well-known cause of attacks of faintness is reflex gastric disturbance, although actual fainting is far less common from this cause than a sense of faintness often accompanied by a little vertigo, such as often precedes an ordinary fainting attack.

I have just given two cases where cardiac failure was simulated by fainting due to some other cause. I will now speak of a case where faintness of cardiac origin was diagnosed and treated as being simply of reflex gastric origin.

Although I did not personally examine the patient, the circumstances were known to me, and I give a probable analysis of the case to illustrate how the principles, here laid down, would apply where—as in this instance—there was myocardial degeneration that could easily be overlooked.

The patient was an elderly gentleman of thoroughly healthy appearance and active in mind and body, and almost the only thing he complained of was a slight passing feeling of faintness or giddiness that, while rather alarming his friends, did not cause him any serious inconvenience. He was noticed to be decidedly breathless when walking too fast, especially uphill; but as it had only gradually increased in degree, and did not trouble him in ordinary exertion he did not think anything of it. The seriousness of his condition was not recognised till, while apparently in perfect health, he one day suddenly had a fainting attack while standing, during the act of micturition, and never recovered consciousness.

He had seen a consultant as to his attacks of faintness, and he had treated them as being simply gastric in origin.

What were the physical signs which might have revealed the presence of myocardial degeneration? From my experience of this type of cardiac failure, I think I shall not be far wrong in suggesting that if the case had been studied in the manner here suggested the following important facts would have been brought out.

First as to the cardiac action. The pulse was probably a good normal pulse for a man of his age, but probably careful observation would have shown that it collapsed a little too readily between the beats, giving it rather a short jerky type. Had it been poor and weak, the able specialist he went to see would certainly have recognised myocardial degeneration as the cause of his faintness. As regards the impulse of the left ventricle, the apex beat may or may not have been palpable. The probability is that there was no cardiac impulse at all.

As regards the right ventricle. The veins of the neck would have been empty and the supra-clavicular fossæ deep. The jugular bulb would almost certainly have been quite indistinguishable owing to emptiness of the vein and absence of pulsation ; or, if after exertion the veins of the neck filled up, as they might do then, there would be practically no pulsation in them except a slight amount of false pulsation. There would be no pulsation to left of the sternum or in the epigastrium, showing weakness and emptiness of the right side of the heart. In addition to these there would be well-marked signs that the amount of blood in active circulation was less than normal—namely, the gastric resonance would be up above the normal level, and quite possibly well into the fifth interspace, and the dullness of the liver and the heart would probably be less than normal.

This is a picture of what such a case might well show; but be it noted there is not a single one of these signs that might not be passed over as being quite unimportant, even if it were observed at all ; and not one of them, except the absence of pulsation in the jugular bulb, that ought not rightly to be passed over as probably being quite unimportant if it occurred *singly* ; but the occurrence of all the signs together, insignificant though they may be, makes a certain and unmistakable picture of dangerous myocardial degeneration—probably fatty and due to disease of the coronary artery supplying the right ventricle and to a less extent the left.

The difficulty of this case lay undoubtedly in the fact that the right ventricle was in the main affected, while the left was fairly strong and well nourished.

His activity of mind and body depended on this fact—that the left ventricle was capable of any ordinary call that he made upon it even in the matter of public speaking and much travelling about. The weakness of the right side showed itself in the very marked dyspnœa that accompanied any overstepping of the limits of what the right ventricle was able to accomplish.

In such a case as this the total amount of blood in circulation would be cut down by the inability of the right ventricle to pass the normal amount through the lungs, and nature's compensatory mechanism would cut down the blood in active circulation to this amount. In such a case as this it is sometimes noticeable that distinct dyspnœa is produced while taking the blood pressure as soon as the pressure on the veins of the arm is great enough to interfere with the supply of blood to the heart (see p. 140). The occurrence of such dyspnœa in this case would have given confirmatory evidence as to myocardial degeneration.

The fatal fainting attack took place, in all probability, because the sudden emptying of the bladder lessened the return of blood to the heart by delaying its return from the abdominal veins, in the manner pointed out many years ago by Sir Lauder Brunton.

Had the diagnosis of myocardial weakness been made, some years might have been added to this patient's life; for in all such cases the caution should be given never to pass water when standing up, but only when sitting or squatting. It is an interesting fact that the natives of India always urinate in this latter attitude; and it is possible that the occurrence, not infrequently, of sudden deaths while micturating standing, did, centuries ago, give rise to the superstitious belief that to do so was offensive to the Ruler of the Universe, and hence led to the adoption of a more humble attitude.

ANGINAL PAIN

There are diagnostic difficulties associated with the occurrence of anginal pain; but these are mainly as regards the differentiation between the pain arising from a temporary spasm of the arteries due to reflex disturbance—the so-called 'vasomotor angina,' and that due to organic disease of the heart or the arteries. There is little if any risk here of mistaking a true cardiac pain for one merely of gastric origin.

As to the diagnosis of angina from other forms of pain little need be said here, as the subject is fully dealt with in the many books on the heart which exist.

The diagnosis of true angina rests upon similar observations to those already referred to—namely, especially upon observing the strength of the heart as judged by physical examination and by noting the amount of exertion the patient can take without bringing on an attack of pain.

When noting the strength of the heart it must not be forgotten that true angina pectoris can occur in a patient with a strong heart, when the work it has to do is sufficiently increased by high arterial resistance and loss of elasticity of the first part of the aorta, just as readily as it can occur in a heart with weakened muscle, which is striving to carry on the circulation under normal arterial conditions.

In illustration of this subject it may be of interest to give two striking cases of reflex angina, and in both the cause was the irritability of the colon, which results in the condition which I have called 'colon dyspepsia,' and which would be less correctly embraced under the well-known term, mucous colitis.

The following is a case of what might be called vasomotor angina, which might very easily be mistaken for true angina, due to cardiac failure. The patient was a lady aged forty-two, who for some years had had occasional attacks of pain in the chest, usually brought on by exertion. The pain was usually located just over the heart at about the level of the fifth and sixth ribs. Sometimes it would be felt to run up the left side of the neck as far as the angle of the jaw, and for five minutes or so it would be like a severe neuralgia. In the more severe attacks it started in the left sub-clavicular region about the coracoid process, and would run up over the shoulder and right down the arm to the thumb, the pain being mainly along a narrow area an inch or so wide, running down the front of the upper arm and the radial border of the forearm to the thumb. The pain was very severe under the thumbnail. In a very severe attack the only other place where she felt pain was under the nail of the left great toe. The pain was often so severe here as to be almost unbearable. The toe, too, was often so tender of an evening that she could not even wear soft felt slippers. The pain was usually accompanied by a sense of suffocation which she describes as ‘ a terrible gasping sensation, which made her feel as if she were dying.’ She often also had palpitation with the attacks. When the attacks were severe, the feeling that death was impending was very strong, and sometimes for many evenings in succession she would sit up till quite late at night because she was afraid to go to sleep, owing to the uncertainty she felt as to whether she would be alive in the morning.

It seemed quite certain that these attacks were associated with cardiac weakness; for hurrying upstairs would always bring on pain, and anything lowering the nerve tone would have the same effect. During the four months prior to my seeing her, she had had much domestic anxiety and physical strain and bereavement, in that she personally nursed a child and her husband, both of whom died within a month or two of each other. The strain of this time had greatly increased the frequency of the attacks of pain, and latterly they had been as frequent as four and five times a day.

Their cardiac nature was also suggested by the fact that nitroglycerine would relieve them in ten or fifteen minutes, and that if nothing was taken they would last some hours.

Another point was that when the sense of suffocation was severe she could sometimes by a single ‘ long breath ’ get relief for five minutes. More often, and especially of late, she cannot do this, and ‘ the breath seems to stop half-way.’

A point in the history which is not quite characteristic of pure cardiac angina is the fact that she was very apt to have an attack on going to bed at night, lasting a quarter of an hour or so. These were often associated with severe abdominal pain and a sense of a band of iron right across the abdomen just at or above the umbilicus.

She also had occasionally severe attacks of prostration associated with symptoms of Reynaud’s disease.

This is an account of a typical one.

One Saturday last summer a gastric attack began just before she started on a journey to London. It began with an intense distaste for food and she could not even drink water.

Nevertheless, she persevered with her journey, spent the whole of Sunday in bed, and got back to Birmingham at 4.40 P.M. Monday. During the whole of that time she asserts that she touched neither food nor water. She says also that the hands and feet were blue-grey and dead from 9 A.M. on the Sunday till the Monday evening. She also had a very severe headache. The blueness of the hands is a feature of every severe attack.

On examining her, the heart was found to be apparently normal in size and action, except that the right ventricular sounds were a trifle louder than normal. The venous circulation, too, was normal, and there was nothing to suggest myocardial failure. On examining the abdomen it was found that the cæcum was markedly hard and tender, as was also the sigmoid flexure; but the somewhat severe pain which moderately firm palpation of the cæcum caused had a most interesting result. It immediately produced a most marked flushing of the face and a little blueness of the hands, accompanied by one of her typical attacks of anginal pain in the heart and down the left arm. In connection with this tenderness of the cæcum, and the fact that pain there could produce an attack of angina, she said that the right side was always tender when she had an attack, and that the attack seemed to start there and then go to her heart.

At a subsequent visit the transverse colon was found to be in a state of extreme expansile tone throughout its entire length, and was quite visible right across the abdomen. It was expanded to about the diameter of a lady's wrist, and was quite hard to the touch and extremely tender. It was this hardening up of the transverse colon that gave the patient the sensation she described as severe pain accompanied by the feeling of having an iron band across the abdomen. This, then, was a case of extremely irritable colon. It might well be called colon dyspepsia, for the term mucous colitis was hardly applicable —there being no excess of mucus in the stools.

This patient's condition was very greatly improved by the adequate treatment of her colon.

It was not a case where the pain showed the presence of cardiac weakness. The case was one where the powerful afferent impulses from the extremely irritable colon—which ought to have caused very severe abdominal pain (but did not)—expended all their energy upon the vasomotor mechanism. On reaching the cord through the sympathetic nerves from the colon, instead of getting into the sensory tracts and causing pain, they, as it were, lost their way and got into the vasomotor tracts, with the result that they caused so severe an arterial spasm that the heart was overtaxed and cardiac pain resulted. The deadness and blueness of the hands which accompanied the pain was another evidence of the arterial spasm. The severity of the vasomotor disturbance is shown by such attacks as the one recorded when the whole of both hands and feet remained dead and grey for twenty-four hours.

The part played in this case by the reflex mechanism is shown by the fact that any circumstances which were calculated to heighten the excitability of that mechanism were sure to increase the frequency and the severity of her attacks of pain.

Another case of extreme interest is the following :—

The patient was a gentleman of fifty-five, whose pleasure, as well as the support of his household, was more or less closely associated with the following of the hounds in the hunting-field.

He was sent to me by his doctor because of the somewhat frequent recurrence, of late, of severe pain in his chest, accompanied by a sense of weight and tightness across the chest, but not by breathlessness or a sense of impending death as in the last case. Another point of some importance was that nerve worry and strain was far more likely to bring on the pain that physical exertion. When the pain was liable to occur it would often come on, when hunting, with the first gallop of the day, and his custom was to get off, take a dose of nitroglycerine till the pain subsided, and then get on again and continue hunting, which he found he could do even throughout the whole day, and on a pulling horse, without any return of the pain. If he did not take the nitroglycerine, the pain would go off, though less quickly, but would be apt to recur, after remounting, the next time he had a gallop. Another instance of the mode of occurrence and disappearance of the pain is interesting and instructive. It came on one day when he was walking to the post, but as he had to catch it he—to use his own expression—'braced his shoulders back and clenched his arms,' and by the time he got to the post the pain had gone.

The examination of the heart suggested a certain amount of cardiac weakness, in that the cardiac area was small and the gastric resonance a little high and the beat of the heart and its sounds rather feeble. The blood pressure was 120 mm., and the volume of the pulse very good. The venous pulsation, too, was good in tone. No signs of definite cardiac weakness on exertion could be detected. For instance, going uphill to church on a Sunday would bring on the pain, but climbing hills when on a holiday would not (see fig. 19).

The strength of a chain being its weakest link, it was evident that the anginal attacks could not primarily be due to cardiac weakness or they would *always* come on when the heart was taxed beyond a certain point. An interesting point about the case was that on first starting out, galloping on horseback was almost certain to bring on an attack, whereas later in the day it did not. Also the attacks of pain often came on suddenly and without cause, especially at night. Both these last two facts point to the possibility of the attacks arising reflexly from irritability of the colon, and especially the transverse colon, to judge from the fact that shaking during the act of galloping was apt to bring it on. The fact that the first gallop of the day brought it on, whereas, later on, galloping did not, is quite characteristic of this mode of onset. The shaking of the transverse colon caused a muscular spasm at first; but, later on, when the bowel got used to the movement, no spasm resulted. The cessation of pain in the bowel (or of reflex disturbance occurring in place of pain), in spite of a continuance of the movement that first caused it, is a common phenomenon in cases where the transverse colon is irritable. In this case the examination of the abdomen showed marked tenderness of the colon, and, further, a history of occasional pain in the groin was obtained. Moreover, the man was almost cured of his cardiac symptoms by intestinal antiseptics and a diet adapted to his colon dyspepsia.

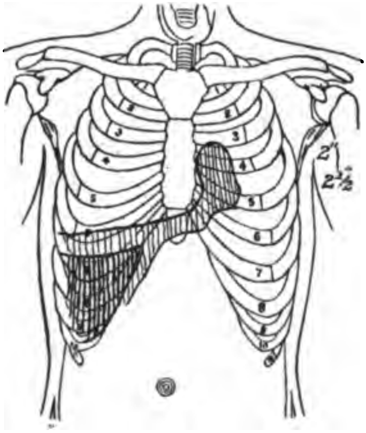


FIG. 19.

Colonel F., aged fifty-five, January 1911. Vasomotor angina. Cardiac area rather small, and gastric resonance high.

Another interesting point was this. Intentional rough handling of the tender cæcum or sigmoid flexure not only caused some of the pain in the chest from which he was apt to suffer as well as pain in the abdomen, but it also caused some blanching of the fingers and hands (a tendency to ' deadness '), thus showing that definite arterial spasm could be produced at will by irritation of his colon, and giving strong evidence in favour of his attacks being due to a vasomotor disturbance, whose source of origin was the irritable colon. The diagnosis of such a case as this is not difficult if the evidence be properly weighed, as was done by his doctor who sent him to me, as a case of false and not true angina.

Other cases of false angina might be given, but these must suffice.

Where there is severe pain in the upper part of the abdomen it is sometimes not easy at first to say whether it be of abdominal or cardiac origin.

THE DIAGNOSTIC VALUE OF VENOUS PHENOMENA

In middle and later life the venous phenomena which are due to the presence of tricuspid regurgitation, or which characterise its absence when it might reasonably be expected to be present, are of very great clinical importance.

Although the study of the veins is of importance in the moderately distensible heart of adult life, the information they give is of greater value where the heart is too rigid to admit of dilatation, and the condition of the veins is of yet greater value in cases where the muscular failure of the heart is so considerable that it is unable to maintain a condition of venous plethora at all.

In the present connection the phenomena of venous plethora only will first be noted.

The various points from which important information can be gained will now be summarised.

1. When first observing the condition of the veins of the neck a distinction must be made between the condition of the great veins (the innominates and the subclavians) and that of the jugular and other neck veins which are on the distal side of the valves which guard the veins opening into the subclavians and the innominates. There is an important reason for this distinction, because the great veins form

practically a part of the auricular reservoir for supplying the right ventricle, and the subclavian valves may therefore be regarded as cardiac valves—so important is their action in the circulation of the blood. These great veins, and the auricle constitute what is practically the compensation reservoir of supply for the right ventricle, and the blood in it is always kept under pressure by the tonic contraction of the muscles in the walls of the veins and of the auricle. There is no evidence of any true relaxation of the walls of the auricle during its diastole, and the same is true of the right ventricle. During the phase of the diastole, which I have called the 'relaxation phase,' there is still tonic activity on the part of the cardiac muscle, and the term relaxation is only a relative one. In this the cardiac muscle is like other muscles of the body which have their periods of activity and of quiescence, but during their quiescence still retain a certain measure of tonic, as distinct from true contractile, activity. This point is well brought out, and in my opinion definitely proved by the careful study of the venous and cardiac tracings which form the subject of Essay XVII, p. 489. This case shows that the blood in the reservoir is under pressure and that waves can be reflected backwards and forwards between the subclavian valves and the wall of the right ventricle during its relaxation period in a manner that would not be possible were the walls of the veins or the ventricle really lax in any absolute sense. Fortunately, nature has arranged that in one spot this venous reservoir comes within our reach for purposes of observation—namely, at the spot where the external jugular vein enters the subclavian. The valves which guard this vein are situated some little distance from the subclavian, and just come within reach of the finger if pressed down behind the clavicle. In this way we can by palpation measure the height of the blood pressure in the auriculo-venous reservoir and thus gain very important information as to the power which is being developed by the right ventricle, for its beat can be plainly felt and we can also judge by the feel of the vein under the finger as to the presence or absence of over-distension and of the strong beat due to true tricuspid regurgitation. This portion of the jugular vein has been christened by Sir J. Mackenzie 'the jugular bulb,' in consequence of the sensation which it gives to the finger when the pressure in the auriculo-venous reservoir is raised above the normal, and it seems a very apposite term.

The examination of the jugular bulb is as important in judging of the work of the right ventricle as the examination of the radial pulse is in the case of the left ventricle; and the information which it gives, as to the power of the cardiac muscle, is, in later life, even more reliable than that given by the radial artery. It would be quite suitable to describe the pulsation felt in the jugular bulb as *the* venous pulse.

In addition to the information given by the palpable impulse felt in the jugular bulb, it is also possible to gain some knowledge as to the extent to which the auriculo-venous reservoir is filled with blood, for when the superior cava and its branches are more distended than normal the hollows above the clavicles and in the episternal notch are less evident than when the veins are emptier. When the veins are much over-distended as a result of tricuspid regurgitation the pulsation of the subclavians or innominates may be distinctly visible and palpable behind the clavicles or in the episternal notch. When the veins are emptier than normal (for instance, in severe myocardial weakness), the hollows above the clavicles and in the episternal become more pronounced than normal.

In addition to the actual ventricular contraction, another factor is concerned with the fullness or emptiness of these hollows of which we are speaking, and greatly adds to the importance of carefully noting their condition. For whenever (as a result of cardiac failure in a fairly strong heart) there is venous plethora the abnormal fullness of the lungs and venous system will not only cause a lowering of the diaphragm, but the same abnormal intrathoracic pressure will cause a bulging of the areas above the clavicles. In the same way when, owing to grave myocardial weakness, there is emptiness of the lungs and the venous system, the same negative pressure which causes the diaphragm to rise will also cause an indrawing of the tissues in the supra-clavicular spaces and the episternal notch, and give rise to a deepening of the hollows of which we have spoken. Therefore, we can by palpation of the jugular bulb and by observation of the general fullness above the clavicles gain important information as to the fullness or emptiness of the auriculo-venous reservoir and of the strength of the contraction of the right ventricle. Although the fullness of the venous system and the increase in the strength of the pulsation, here spoken of together, *are* usually associated clinically, it is not necessarily so; and sometimes very considerable fullness of the veins may be associated with a weak ventricular pulse, and when this is so, it gives an important indication as to the state of the circulation, and points to myocardial weakness and often to some sudden embarrassment of a weak ventricle; on the other hand, an abnormally strong jugular pulse may be unaccompanied by any marked over-filling of the general venous system of the thorax.

When dealing with the veins in the distal side of the valves which guard the subclavian and innominate veins, there are two distinct directions in which we have to gain information.

Firstly, Are the veins of normal fullness or are they fuller or less full than normal? and, secondly, Is there any evidence that the venous valves have given way and are allowing a reflux of blood from the auriculo-venous reservoir into them during the ventricular systole?

The degree of fullness of the veins above the valves, while not giving as striking evidence, as to the general condition of the circulation, as is obtained from the reservoir on their proximal side, is yet of importance as confirming the evidence thus obtained; but so far as positive evidence of the early occurrence of cardiac failure is concerned, the evidence is of more value than that given by the auriculo-venous reservoir, because, owing to their accessiblity, the veins give evidence of quite slight changes in the effectiveness of the work of the right ventricle.

So far as concerns the negative evidence given by their emptiness, this is of but little value as compared with that given in other ways, as already described.

As regards true pulsation in the veins of the neck, due to tricuspid regurgitation, it does not at the present time seem possible to speak definitely, so far as the full interpretation of the different types of pulsation is concerned. The kind of pulsation varies much in different cases. Sometimes there is a distinct double beat right up to the angle of the jaw. A good deal remains to be done by the careful record and study of the venous pulse by means of the cardiograph ; for the writer is impressed by the fact that the current interpretation of venous tracings is, to say the least, somewhat inadequate. It is extremely likely that some of the sudden movements seen in the veins, as recorded by the cardiograph are really due to a true contraction of the muscular walls of the veins themselves.

With a fuller knowledge of the meaning of the venous pulse, we shall gain a fuller knowledge of the mode of action of the right side of the heart under the varying conditions which disease induces.

We must now go a little more into detail, with regard to the information we can obtain, by studying the veins of the neck. The subject is, however, somewhat difficult to deal with concisely, owing to the complicated nature of the interrelationships of the various factors concerned, and therefore anything approaching completeness or scientific method must not be expected.

In the first place, a simple table (see pp. 137 and 138) will be given showing the main diagnostic points to be noted in connection with the state of the veins and the probable interpretation of the various groupings of the phenomena observed, and then some rather fuller notes upon the same subjects will be given.

Some of the remarks will be so simple as to be self-evident, whereas the truth of others may appear questionable at first sight, although experience will probably prove their reliability in most instances.

They will be given as a series of aphorisms.

The following remarks illustrate rather more fully some of the points to which attention is drawn in the table.

Fullness of the neck veins, with manifest systolic pulsation in them, suggests a strong right ventricle working against abnormal resistance to the circulation through the lungs. The commonest cause for such interference with the pulmonary circulation is, perhaps, disease of the mitral or aortic valves.

A manifestly excessive carotid pulsation associated with strong pulsation in the neck veins would make the diagnosis

VENOUS PHENOMENA OF CARDIAC FAILURE

No.	Distension of veins of neck	False pulsation	True pulsation	Jugular bulb pulsation	Diagnosis
1	None	None	None	Moderate strength	Normal circulation.
2	None	None	None	Very strong and full	Failing right ventricle where tricuspid regurgitation is entirely downwards to the liver.
3	Some	Some false	None	Normal	Slight right ventricle failure. General dilatation, if present, not enough to cause tricuspid regurgitation. In the heart failure of anæmia in adolescence, slight pneumonia, &c.
4	Some	Well marked	Slight or none	Too full and strong	Some general dilatation of right ventricle and commencing tricuspid regurgitation.
5	Well marked	Present	Some	Too full and strong	Some tricuspid regurgitation.
6	Well marked	Well marked	Some	Too full and strong	Severe embarrassment of right ventricle. Sudden somewhat severe pneumonia or asthmatic attack.
7	Well marked	Little	Well marked	Too full and strong	Considerable tricuspid regurgitation. Considerable degree of failure of left ventricle from valvular disease or overstrain.
8	Well marked	Little or none	Strong	Not palpable	Considerable tricuspid regurgitation with failure of venous valves. Failing compensation with good heart muscle.
9	Well marked	None	Visible but feebly palpable	Feeble	Onset of muscular feebleness complicating tricuspid regurgitation.
10	Moderate	None	Feeble	Feeble	Weaker muscle.
11	Little	None	None	Very weak.	Very weak muscles of right ventricle.
12	No fullness	None	None	None	Considerable fatty degeneration of right ventricle.

VENOUS PHENOMENA OF CARDIAC FAILURE—*continued*

No.	Distension of veins of neck	False pulsation	True pulsation	Jugular bulb pulsation	Diagnosis
13	Empty and hollowness above the clavicle	None	None	Small but moderate strength	Degeneration of left ventricle with moderately strong right ventricle.
14	Empty and hollowness above the clavicle	None	None	None	Dangerously weak heart muscles from intoxication or fatty degeneration.

of aortic regurgitation with failing compensation almost certain. The association of increased cardiac dullness to the right of the sternum and increased pulsation over the right ventricle just to the left of the sternum, with strong venous pulsation, would suggest that the heart was distensible; whereas the absence of right ventricular dilatation under such circumstances would be strong evidence that the heart was relatively rigid.

A well-marked systolic impulse in the neck veins without any evidence of valvular disease would suggest some interference with the pulmonary circulation by some affection of the lungs (emphysema, for instance), or the recent onset of failure of the left ventricle from overstrain, from muscular weakness, or from ineffective action such as is associated with certain types of arythmia.

Where the fullness and systolic pulsation are only moderate in amount, there are many more alternatives than when both are considerable. In such cases, the following generalisation can be made.

Broadly speaking, it can be asserted that muscular weakness of the heart can be diagnosed whenever well-marked cardiac failure is associated with the absence of well-marked pulsation in the neck veins, whether they be full or empty, always provided that the presence of tricuspid regurgitation downwards into the hepatic vein has been also excluded.

This generalisation can also be carried further ; for it is safe to consider that in well-marked heart failure the degree of weakness of the pulsation and of the emptiness of the veins is a measure of the degree of muscular weakness of the right ventricle which is present.

In an extreme case, great dyspnœa may be associated with emptiness of the neck veins, and well-marked hollows above the clavicle and in the episternal notch. Such a condition will point to the presence of well-marked fatty degeneration of the right ventricle. This diagnosis will be confirmed by weakness of the right ventricular heart sounds and the absence of any cardiac impulse just to the left of the sternum or in the epigastrium. If the myocardial weakness involves the left side as well as the right its sounds will be feeble, its impulse weak or absent, and the radial pulse will also give evidence of the fact.

If, on the other hand, the degeneration mainly involves the right side and the left is fairly strong, the apex impulse and sounds, and the radial pulse, may all be fairly strong. Sometimes, indeed, in such a case as this the radial pulse may show a strong wave with a blood pressure as high as 160 mm. of mercury, although the patient may be within a few hours of death.

Moderate fullness of the neck veins and moderate pulsation in a patient with valvular disease would be evidence that compensation was not very unsatisfactory.

In a patient with a dilated left ventricle from overstrain, the meaning of moderate fullness and pulsation of the neck veins would depend largely upon the other phenomena present.

For instance, with a history of increasing breathlessness and a feeble diffuse apex beat, good pulsation in the veins of the neck, when associated with signs of some overaction of the right ventricle, would suggest failure of the left side with compensatory overactivity of the right, and would call for careful stimulation and also for lessening of the heart's load by lowering the blood pressure if there were evidence that an increase in the arterial resistance was a factor in the case.

If, on the other hand, moderate fullness and pulsation of the neck veins were associated with signs of a moderately strong right ventricle and of a normally strong left ventricle, the venous fullness and pulsation might give no special indication for treatment, but be simply a phenomenon that showed the highest level of efficiency to which a weak heart could attain.

In such a case any increase in the feebleness of the heart would be associated with the disappearance of the pulsation, and a return of strength would be accompanied by its reappearance.

This is a point which must be remembered when dealing with the myocardial failure of later life, both as regards diagnosis, prognosis, and treatment; for there are many pitfalls in connection with feeble and absent pulsation in the veins of the neck. In a strong right ventricle with a strong left one, there is sure to be a venous plethora with well-marked pulsation whenever the left ventricle is sufficiently overburdened.

In a weaker right ventricle with a strong left there will be well-marked venous plethora, but with weaker systolic pulsation in them.

In myocardial weakness of both sides of the heart the amount of fullness and pulsation will be proportional to the heart's vigour.

When fullness of the veins is accompanied by little or no pulsation in them it is strongly suggestive of the fact that the right ventricle is weaker than the left.

It has been already pointed out that in a case of considerable muscular feebleness, with empty and non-pulsating veins, a reappearance of fullness and of pulsation in them would be a sign of returning strength. It may not always be so, however, as in the following clinical history. This case seems to show clearly that emptiness of the venous system is, in myocardial weakness, a compensatory phenomenon, and that a weak heart works better when the veins are empty than when they are of normal fullness.

The patient was a lady over eighty years of age, who suffered from high arterial resistance with dilatation of the left ventricle and an amount of heart weakness that, while readily causing breathlessness and fatigue on physical or mental exertion, was not enough to prevent her from going up- and downstairs. In spite of this degree of activity there was in this case compensatory diminution in the amount of blood in circulation. The neck veins were as a rule not at all full, and were in all probability less full than normal, for the compression of the veins caused by the application of the armlet of the sphygmomanometer always caused a sense of breathlessness accompanied by deep, often sighing, inspirations. The venous blood supply to the right heart being so limited that some embarrassment resulted when the venous return from the arm was prevented. In this patient it was thought that relief would be given by lowering the peripheral resistance by means of nitroglycerine, and after a few half-minim doses, there was marked increase in the fullness of the neck veins, showing that more blood was passing through the capillaries, and also the application of the armlet no longer caused dyspnœa, showing an increase in the amount of venous blood available for the supply of the right ventricle; but the patient's condition was made worse. She was more breathless on exertion, and it was evident that the effect of the vaso-dilatation was to supply the heart with more blood than it could properly deal with.

Such a case as this shows how complicated is the problem which we have before us in hearts that are muscularly weak, and especially in those cases where there is high blood pressure and increase in the peripheral resistance in the arteries. This patient still possessed sufficient distensibility of the right ventricle to give useful indications for treatment. Any overstepping of the prescribed limitations as regards exertion was apt to cause the appearance of pulsation over the right ventricle just to the left of the sternum, and, on the other hand, improvement was associated with a diminution in its pulsation and also its size, as was shown by the appearance of full lung resonance just to the left of the sternum in the fourth interspace. In another patient the interpretation of these physical signs might be the reverse of what it was here, so far as the size of the right ventricle is concerned; for the diminution in size, which here showed a return to normal, might in a case with greater myocardial weakness mean a lessening of the heart's power and be accompanied by increased dyspnœa and weakness.

In fact, in this same patient, when she was suffering from an attack of pneumonia, the absence of right ventricular pulsation, combined with this same diminution in the amount of dullness just to the left of the sternum, was a sign of dangerous weakness of the right ventricle (*vide* p. 105).

HOW FAR CAN WE ASCERTAIN THE PART PLAYED BY ARTERIAL RESISTANCE IN CARDIAC FAILURE?

In the cardiac failure of later life increased arterial resistance plays a very important part in consequence of the frequent occurrence of abnormalities of peripheral resistance due to arterial disease or spasm. In any given case, however, it is not at all easy to appraise at its proper value the share which such increased resistance as may be present is taking in bringing about the cardiac failure. In the earlier decades of life this was not so, and it was comparatively easy to say to what degree the cardiac failure in any particular case was due to a pathological increase in the blood pressure.

The fact that the part played by increased peripheral resistance is often an unknown factor in our cardiac problem greatly complicates our diagnosis, whose aim, especially in the cardiac failure of later life, is to ascertain the strength of the heart muscle.

Let us take an instance.

The symptoms evidently, let us suppose, point to heart failure, and the question to be answered is this: Is the failure due to the heart being called upon to do more work than normal? or is the failure due to the heart's inability to do what may be called a normal amount of work because of muscular weakness? In early life the answer to this was easy, because the heart was so distensible that the yielding of its wall (i.e. dilatation) would at once give evidence of any abnormally great intraventricular pressure, and thus show whether the failure was due to excess of work or to muscular weakness.

In later life, however, the problem is complicated by the indistensibility of the heart wall, and therefore, before being able to solve our problem, we must know either what the distensibility of the heart wall is or else what the arterial resistance is against which the heart is working, before we can decide as to the strength of the heart muscle. In every case, we want to know what the strength of the heart muscle is and what the arterial resistance is against which it has to work, and until we do know this, we cannot properly treat the case or give a reasonably accurate prognosis. We are too apt when we meet with a case of cardiac failure just to try cardiac tonics or vascular relaxants without really having taken the mental exertion of analysing the symptoms and signs and weighing up the evidence for and against muscular weakness of the heart wall. The distinction between a strong heart and a weak one is fairly easily made, even in the absence of cardiac dilatation, because we have a measure of its strength in the loudness of the sounds and the amount of venous engorgement and pulsation. The absence of these phenomena will indicate that myocardial weakness plays an important part in the cardiac failure.

We shall therefore now go into the difficult question of how far it is possible to ascertain what part increased arterial resistance plays in any particular case of heart failure.

VALUE OF THE RADIAL PULSE IN THE DIAGNOSIS OF CARDIAC FAILURE IN LATER LIFE

As has already been stated, the condition of the pulse is often a most unreliable guide in dealing with the heart failure of later life, except in the case of marked weakness. There are two good reasons for this. Firstly, unless the character of the pulse prior to the onset of the cardiac failure is known, it is almost impossible to be sure that a normal or moderately strong pulse is not after all a greatly weakened high pressure pulse, and therefore a sign of greatly weakened heart.

Secondly, it is not at all easy to tell from the character of the radial pulse what the condition of the right ventricle is. It is not very uncommon to find cases with a dangerously weak right ventricle in whom the radial pulse is fairly strong and good.

The main problem from a diagnostic point of view is however, associated with the question, How can we ascertain the amount of work which the heart is doing so far as the arteries are concerned ?

It is not enough to recognise that the pulse tension is high—as judged by the finger—or that the blood pressure is high—as judged by the sphygmomanometer—unless we are able at the same time to say from our previous knowledge of the case that there has been a rise in the blood pressure and that therefore there is reason to expect that the attack of cardiac failure which we have before us is probably due to the extra load thus thrown upon the heart. If we have not this previous knowledge as to the condition of the circulation, the blood pressure, high though it be, may yet be lower than what is normal for the patient, and the cardiac failure may be more due to myocardial weakness than to abnormal peripheral resistance. It is quite possible that in such a case the right treatment might be to try to raise a blood pressure already at the level of 170 mm. or 180 mm., and that the proper nutrition of the heart muscle would not be restored till this was done.

There is a question which must be asked in this connection —namely, Is it not likely that we may occasionally meet with cases of high blood pressure which are compensatory in their nature? Cases, say, where there is so much narrowing of the coronary arteries (the result of atheroma) that an adequate supply of blood will not pass through them at the ordinary pressure, and where in consequence the blood pressure has to be abnormally raised by vasomotor activity in order to ensure the proper nutrition of the heart muscle. In such a case as this it is easy to see how disaster would follow the attempt to lower further a high blood pressure which had, in consequence of the onset of myocardial failure, already dropped below the safe level.

The question illustrates the difficulty of the problem before a man who sees a case of cardiac failure in later life for the first time and has to interpret the phenomena observable in the radial pulse.

For the solution of this problem some assistance can, I think, be gained by the sphygmomanometer if, in addition to noting the effect of the rise in pressure upon the pulse alone, attention be also directed to the range of movement of the mercury column at the various pressures to which the artery is exposed, or, better still, by noting the range of movement of some form of oscillometer; for by this means we can tell whether the height of the blood pressure is determined by the strength of the heart's contraction alone, or whether the heart is being forced to beat more powerfully than would otherwise be the case owing to contraction of the peripheral vessels.

To take the latter case first.

If there be an abnormal amount of contraction of the peripheral vessels and the heart is strong enough to respond to it, we shall find that the range of oscillation remains at its maximum almost up to the point which we call the systolic pressure, when the artery is so compressed that the blood can no longer be forced past the point of compression. Thus supposing the range of oscillation is 2 mm. of mercury and the systolic pressure is 150 mm., we shall find that at 146 or 148 the range is still 2 mm. of mercury, and at 150 it suddenly drops to 1, and the pulse is no longer felt at the wrist, showing that the heart is strong enough to keep up a full volume of blood in the artery as long as it can force any blood at all past the compressed point. If, on the other hand, the range of movement gradually lessens—say, at 130 it is 2 mm., at 138 1½ mm., at 144, 1 mm., at 150, 1 mm.—we should, I believe, be justified in saying that the state of the arteries was not the controlling factor in determining the height of the blood pressure. In the normal heart with normal arteries this gradual subsidence in the range of oscillation is the rule.

If, therefore, we found a pulse with a systolic pressure of 160 or 170 mm., with this gradual subsidence in the range of oscillation, we should be justified in suspecting that it was a case where the blood pressure was below what was normal for the individual and that some myocardial weakness was present. In such a case, we should be prepared to see a rise in the measured blood pressure as soon as the heart regained its strength ; moreover, in such a case, we should be careful as to the administration of nitrites for fear of still further impairing the nutrition of the cardiac muscle by further lowering a blood pressure that was already abnormally low. If, on the other hand, we found that the full range of oscillations continued close up to the maximum pressure that the artery would stand, we should expect to see benefit result from the use of nitrites, and should continue our efforts to lower the blood pressure until we found that the type of oscillation changed, thus showing that the heart was no longer working against a resistance due to contracted peripheral vessels.

Assistance will also be given by watching the oscillations in the possible cases of compensatory high blood pressure, already spoken of; for in such a case as this a lowering of the blood pressure would soon make it clear that there was no real interference with the circulation by pathological contraction or rigidity of the arteries. The study of the oscillations of the pulse would soon show the uselessness of trying to lower the blood pressure in such a case as this.

These are only given as tentative suggestions, because the writer has not studied the pulse by means of the oscillometer for more than a few months—a time too short for anything approaching certainty in so complex a question, and too short also for bringing forward many cases in support of the suggestions here given.

Other illustrations may be given of the fact that in these cases of myocardial weakness phenomena connected with the arteries may have the reverse meaning to that which they have in strong hearts. Take for instance the subject of the blood pressure in cases where the peripheral resistance is increased. A patient is perhaps seen with a hard pulse and a blood pressure of 145 mm. or so. There are signs of a labouring ventricle—for the left ventricular sounds are too short and sharp and the heart is, moreover, irregular—an extra systole with a following pause occurring every eighth or tenth beat for a time with intervals of greater regularity. After endeavouring for many weeks, by means of drugs of the salicylate class and by diet, to lessen the amount of the peripheral resistance, and also by tonics and rest to improve the strength and nutrition of the heart, it was somewhat surprising to find that the blood pressure which formerly stood somewhere about 145 mm. was now 170 mm. or more. This observation by itself might have been disappointing, but it was qualified by the fact that the apex beat was stronger and more localised; the heart sounds also were quieter, less jerky, and more normal in character, suggesting greatly increased strength, so far as the left ventricle was concerned. The pulse, too, was more regular, and the patient rather stronger and less easily tired. The pulse, moreover, felt softer to the finger, although the blood pressure was higher. This was apparently due to the fact that the pulse was less tense between the beats, and emptied in a more normal manner. Here the rise in blood pressure was doubtless the result of increased cardiac strength due to improved nutrition, and was therefore a good sign and not a bad one, as it would have been had it occurred in a heart that was muscularly strong.

The illustrations here given are sufficient to show how little we really know as to the proper reading of the arterial pulse in cases of heart failure.

Even in early life the condition of the pulse may be misleading. For instance, it is probable that in Graves's disease the arterial dilatation is primary, and the overaction of the heart only a result of the arterial change.

This whole subject cannot be adequately dealt with until our knowledge has been considerably increased by the careful study of the range of oscillations of the pulse, and in other ways.

HOW FAR CAN WE ASCERTAIN THE DEGREE OF RIGIDITY OF THE HEART WALL ?

When speaking of the problem which faces us in dealing with heart failure in adult and later life mention was made of the fact that we have an equation with three unknown quantities—namely, strength of heart muscle, arterial resistance against which the heart is working, and the resisting power of the heart wall, and to solve the equation we must ascertain the value of two out of the three unknown quantities.

The question we will now try to answer is : Have we any means of estimating the resisting power of the cardiac wall ?

In certain cases it is easy. For instance, where we find heart failure accompanied by distended veins or enlarged liver with a strong pulsation in the jugular bulb with commencing œdema of the ankles associated with a heart showing no evidences of dilatation, we shall be safe in saying that the cardiac wall possesses abnormal powers of resistance to dilatation. In such a case the cardiac strength is good, and therefore the cause of failure must be sought for elsewhere. The value of two of our three unknown quantities are ascertained, and we can therefore find the third, as follows:—

The failure may be due to valvular disease or some other easily recognisable cause, or it may be due to some condition such as adherent pericardium hampering the action of the heart. These points can be decided with a fair degree of certainty. In the absence of any such cause as these, we can in such a case feel reasonably certain that the arterial system is to blame, and in the absence of any signs of abnormal resistance in such arteries as the radial, we must suspect rigidity of the first part of the aorta and search for signs of this condition.

Again, in a condition such as one of the forms of valvular disease, which is usually associated with dilatation and hypertrophy, the absence of enlargement, if associated with the signs of good muscular strength, would also justify the diagnosis of an abnormally rigid heart wall (*vide* p. 483).

In cases, however, where the heart was weak as well as being either of normal size, or small, the question as to the presence of abnormal rigidity would not be so easily answered. In a case of cardiac failure accompanied by evident signs of muscular weakness, the occurrence of attacks of angina in the absence of evidences of increased arterial resistance would suggest that the heart wall was unduly resistant, and by its powers of mechanical elasticity was apt to draw into the left ventricle a larger amount of blood than the heart muscle had the power of expelling. In such a case as this nitrites would have to be used with caution, because by increasing the permeability of the capillaries and therefore the fullness of the veins, they would increase the risk of the heart overfilling itself (*vide* p. 140). In many cases with weak heart muscle it is not at all easy, if not impossible, to form any reliable estimate as to the degree of rigidity of the heart wall. In this connection attention may be drawn to the following line of argument. A class of case described was at p. 87 where heart failure could clearly be seen to occur in a strong heart with resistant walls, because venous plethora and loud heart sounds were unaccompanied by any dilatation. A clinical group, therefore, exists in which heart failure without enlargement occurs, accompanied by venous plethora and loud heart sounds. But any clinical observer can also convince himself of the fact that cases are often met with where heart failure without enlargement is accompanied by *emptiness of the veins* and weak heart sounds. The recognition of these two types of case as clinical certainties forces upon us a startling and disconcerting fact—namely, that cases of an intermediate nature must sometimes be met with, and that therefore we must sometimes come across cases of moderately rigid hearts with failing heart muscle, where well-marked heart failure occurs, associated with a heart of normal size with normal fullness of the veins and sounds of normal loudness.

In view of the fact that the radial pulse may be quite unreliable for purposes of estimating heart failure, we are face to face with the uncomfortable fact that we may occasionally meet with a case of heart failure where we can gain no help at all from physical examination, because the heart appears quite normal, and we must therefore depend entirely upon symptoms. The analysis of such a case would be as follows:—

1. Abnormal arterial resistance accompanied by sufficient muscular failure to reduce the high tension pulse to one of apparently normal character.

2. A muscularly weak and rigid left ventricle only able to respond to this increased resistance by developing a normal (and therefore inadequate) intraventricular pressure. A weak right ventricle also only able to develop a normal and therefore inadequate pressure, and the veins would therefore have a normal degree of fullness.

Such a heart might improve in one of two ways. Either it might gain a little strength with the result that the sounds would become abnormally loud and the veins overfull, or else if the left ventricle could compensatorily diminish the degree to which it filled itself, the heart sounds would remain of the same loudness, but the blood volume would diminish, the veins become more empty, the diaphragm rise, and the liver dullness diminish. On the occurrence of this latter sequence of phenomena, the heart would once more be able to carry on the circulation fairly well, and the dyspnœa on exertion would diminish to some extent.

What has just been said points to one answer to the question: How can we recognise the presence of rigidity of the heart wall in a very weak heart? We can say this. When we find evidences of decided weakness of the heart muscle (breathlessness, faintness, anginal pain on exertion, leg pains on walking, &c.) associated with a heart of normal size (and not smaller than normal), and with signs that the thoracic viscera contain the normal amount of blood (i.e. that there is no compensatory diminution in the volume of blood in circulation proportionally to the muscular weakness), we are justified in assuming rigidity of the heart wall, and if we are wise, we shall caution the patient, realising that he is running a grave risk of sudden death.

This question of the degree of rigidity of the heart wall is one of much practical importance in later life, and is often one of considerable difficulty. We often meet with hearts that have not dilated under conditions of overstrain that might reasonably have been expected to produce dilatation. The question which has then to be answered is this: Are we dealing with a heart with fair strength and an unduly rigid wall, or are we dealing with a heart with walls of normal rigidity, but with such a deficiency of muscular power that it is unable to develop enough force to over-distend them? If the former, it will be a 'strong heart with rigid walls,' and we have already shown the points whereby such a heart can be recognised. If the latter, it will be a case of weak heart without enlargement, and we must look for signs of weakness and of emptiness of the vascular system—along the lines already given.

EVIDENCES OF RIGIDITY IN CASES OF VALVULAR DISEASE

The principles already laid down apply in this case also, for if we have an amount of dilatation and hypertrophy less than that which might reasonably be expected from the amount of valvular regurgitation present, we are justified in diagnosing undue rigidity of the heart wall, provided that the heart does not show signs of marked muscular weakness. In such a case as this, pronounced pallor will accompany such indistensibility, as is pointed out in the essay dealing with that subject (see p. 450).

As regards the means of judging as to the amount of mitral regurgitation by the extent to which the murmur is conducted down the spine (*vide* Essay, p. 442), and of aortic regurgitation by the amount of pulsation of the carotid arteries (*vide* p. 347), we must be able to form some idea as to the amount of regurgitation before we can say that dilatation and hypertrophy are, or are not, adequate.

There are one or two minor points in connection with the examination of the heart which had better be referred to, although they are but common-sense points, known to every clinician.

When dealing with an area of cardiac dullness that is apparently of less than the normal size, we must make certain, firstly, that the diminution is not due to simple over-lapping by lungs more voluminous than usual, and, secondly, granted that the lungs are, to all appearance normal, we must make sure that there is no evident reason, other than its want of its proper fullness and rigidity, for the heart falling back, away from the chest wall. The variation noticeable in the mobility of the heart in different healthy individuals must not be forgotten.

The possibility also of shrinkage of a portion of one lung as the result of disease, with such a measure of recovery as to make the evidences of it very slight, must not be forgotten, for this might either cause falling back of the heart or else possibly the simulation of enlargement by its being less covered than normally by the lungs.

Also as regards the recognition of enlargement, we must be careful to make our examination of the heart thorough, or we may be caught napping, as the able general practitioner was, who brought the patient whose case is detailed at p. 374 to see me. In this case the practitioner was impressed by the lady's breathlessness on exertion, and by the fact that her apex beat was in the axilla, and diagnosed cardiac failure and dilatation; but, through not percussing out the heart, failed to notice that the right border of the heart was well to the left of the sternum, and that the heart was not really as much enlarged as it appeared to be, but was simply displaced into the left axilla by the gradual and unobserved collapse of the left lung, and the crossing of the right one, which resulted from an almost symptomless but destructive form of tuberculosis.

Or take another case, such as that mentioned at p. 365, where a man with mitral stenosis has an apex beat in the seventh interspace well external to the nipple line. Extreme dilatation of the left ventricle would naturally be diagnosed: but a careful examination of the chest would show that the diaphragm was very low, and that the pulmonary second sound was heard best at about the level of the fourth rib, instead of in the second interspace. Also that the area of cardiac dullness was lower than natural—in other words, this was simply a case of downward displacement of the heart. The displacement was confirmed by a radiograph (as is shown in Plate VI). The patient had suffered from a greatly obstructed mitral valve for twenty years, and the chronic venous distension of long standing had through persistent over-filling of the heart and vessels in the thorax, brought about a downward displacement of the diaphragm, which naturally caused a dropping of the heart also.

Another simple point in connection with the examination of the heart is this. In muscular weakness we have to depend a good deal upon the degree of feebleness of the cardiac sounds, therefore we shall have to look very carefully for the presence of any conditions other than cardiac weakness that may modify the loudness of the heart sounds. The presence of emphysema and its amount must be carefully noted as well as the amount to which the heart is covered by lung when there is no emphysema. The amount of fat on the chest wall must also be noted and its effect estimated.

As regards the evidence given by the size of the liver dullness, we must remember that causes other than cardiac failure will lead to its diminution, as is described in Essay III. Other causes for diminution in the size of the liver dullness must therefore be excluded before we lay stress upon that diminution in connection with cardiac diagnosis. Similarly, where there is no diminution there may be a history, which would suggest cirrhosis, or something else, that would render unlikely the changes which we have associated with heart failure.

While speaking of the liver, it may be well once more to draw attention to a somewhat new and important observation —namely, that percussion is not always a reliable guide to the actual size of the liver, and especially for the type of enlargement which is associated with heart failure where the right ventricle is fairly strong. As is pointed out (pp. 24 and 192), tense liver tissue appears to possess to an abnormal degree the power of conducting percussion and sound vibrations, and thus a resonant note may be obtained through an abnormal thickness of liver tissue.

It is therefore quite possible in a somewhat hasty examination to miss the fact that the liver is enlarged. In such a case, however, it is always possible to feel the edge, and it is no unusual thing to find that there is a fully resonant note for one, two, or even three inches above the level at which the edge is felt.

While at work on this section, the writer saw a case where in typhoid enlargement of the liver there was a good resonant note for two inches or more above the level at which the edge was felt.

Difficulties in Diagnosis due to one Ventricle being Weak while the other is Strong

We now have to discuss a diagnostic difficulty which is associated with one of the most marked differences between the heart in early and in later life—namely, the fact that, whereas in early life the common causes of myocardial weakness are general causes, such as the poisons of infective disease, the commonest causes of myocardial weakness in later life are local and are due to interference with the blood supply to the heart. This being the case, we are prepared for the fact that one of the two coronary arteries may be more interfered with by disease than the other, and that therefore the nutrition of one ventricle may suffer more than the other.

In all our cases of myocardial weakness, therefore, in later life, we shall do well, in studying the symptoms and physical signs, to separate those which mainly depend upon the right ventricle from those which concern the left, in order that we may be able to form a judgment as to whether the patient's symptoms are due to the failure of one ventricle more than the other, or whether both are equally affected.

Firstly, as far as regards the general symptoms. Breathlessness is, as already pointed out, apt to be the prominent symptom in cases where the right ventricle is weak; whereas when the left is the weak one, faintness and weariness and general weakness are more prominent. Faintness is of course apt to occur when the right ventricle is weak, owing to its inability to send on the proper amount of blood through the lungs for the full supply of the left side of the heart.

But the fainting attacks in weak right ventricle are less frequent than in the case of the left, and I think it will be found that frequent 'faint feelings' and slight attacks of vertigo of cardiac origin may be accepted as showing myocardial failure of the left ventricle.

The physical signs need not be gone into fully, for they have been already dealt with *seriatim*; but it must, however, be remembered that a strong right ventricle may be associated with a weak left one, or vice versa, and we may, as in the clinical history shortly to be given, be surprised at a patient who usually has a good pulse dying in a fainting attack.

With a strong right ventricle there is more apt to be fullness of the veins; but I think it will be found that whenever there is marked weakness of one ventricle—no matter which—the characteristic compensatory diminution in the amount of blood in active circulation does take place.

To illustrate the subject, I propose to give one or two clinical histories.

The first case is that of a woman aged sixty-one, who was admitted to the hospital for breathlessness. She had been short of breath for several years, and of late had been gradually getting worse. Recently, she had rapidly got weaker, and the day before admission, she had a serious fainting attack—the first of the kind she had had.

There was no history of any previous illnesses, and her present condition had developed gradually. Her state on admission was as follows: A rather stout woman, very pale, not breathless when lying in bed with a moderately high bed rest, but breathless on exertion and unable to lie flat in bed. There was no dropsy and no cyanosis.

The pulse was decidedly small but of good strength, and was of the strikingly collapsing character usually associated with aortic regurgitation. Although its volume was small it came up under the finger with suddenness and force and subsided again with equal suddenness, leaving the artery empty to the touch between the heart beats. On testing it with the Riva Rocci sphygmomanometer, it was found that the pulse wave would withstand a pressure of nearly 160 mm. of mercury. On testing the collapsibility of the pulse when the arm was raised or lowered, it was found that elevation of the arm did not, as in aortic regurgitation, increase the collapsing character of the pulse. On looking at the neck there was not only no pulsation of the veins, but there was a hollowing above the clavicles which suggested undue emptiness of the vessels.

As regards the heart there was no visible or palpable impulse anywhere, and the cardiac area was small—decidedly less than normal—and the liver was not enlarged. The cardiac sounds were decidedly weak and there was no murmur.

The lungs and other organs appeared quite healthy. This patient was evidently suffering from weakness of the heart muscle of a very severe degree, for the heart had not sufficient strength to keep the superior cava and neck veins distended and pulsating.

The diagnosis was heart weakness due to fatty degeneration—the result probably of diseased coronary arteries. A more complete analysis of the symptoms would have led to an even fuller diagnosis; but the importance of the collapsing pulse, and the systolic pressure of 160 mm., coupled with the empty veins, was not recognised till after the post-mortem revealed the true nature of the case.

The patient was given small doses of strophanthus and strychnine, and seemed to be improving, when suddenly she had another fainting attack five days after admission, and died a few hours later in a second attack. The earlier of the two attacks came on at 4 P.M. There was marked dyspnœa and cyanosis with pain over the heart and left chest, and the pulse was almost imperceptible. She rallied somewhat, but became worse an hour later. In this second attack she suddenly sat up in bed, and then fell back dead. At the post-mortem there was found to be fatty degeneration of the right ventricle, due to atheroma blocking its coronary artery. The condition of the muscular wall of the left ventricle was good, and the coronary artery supplying it was not interfered with by disease.

Here, then, was the explanation of the small powerful collapsing pulse.

The right side of the heart was too feeble to force the proper amount of blood through the lungs, and, as the condition had evidently developed slowly, the amount of blood in circulation had presumably diminished *pari passu* with the diminution in the strength of the right ventricle, thus leading to no venous plethora, as might have been expected had the failure of the right ventricle been less gradual.

The left ventricle was therefore underfilled at each beat, and, in order to get as large an amount as possible into the capillaries of the brain and elsewhere, the blood was thrown out of the ventricle suddenly and with force, the arteries being kept as contracted as possible.

The rapid collapse of the pulse wave and the emptiness of the artery between the beats were due to the abnormally small volume of blood causing the pulse wave.

This case illustrates well the main signs upon which diagnosis of a fatty right ventricle with a fairly strong left ventricle are to be based—namely, great dyspnœa and weak right ventricular sounds, coupled with absence of well-marked venous pulsation in the neck in combination with a regular and not very rapid pulse of small volume but fair strength. Where some amount of arterio-sclerosis is present, the rapid and great collapse of the pulse may not be noticeable; but where present, as in the case here noted, its diagnostic importance is great.

The prognosis, it is needless to add, is in such a case extremely grave.

The following case is one where the diagnosis of fatty or fibroid degeneration involving mainly the right ventricle was, in the writer's opinion, justified by the symptoms, although it was not verified by a post-mortem, as the patient recovered sufficiently to go home. The patient was a jobbing gardener, sixty years of age, who was suffering from emphysema, cardiac weakness, and some nephritis.

His lungs were not in a very bad condition, and his nephritis did not seem to be causing much trouble.

His main complaint was breathlessness. He had been ailing eight years—at first mainly with cough, due probably to chronic bronchitis—and for years he had had dyspnœa on exertion. Within the last six weeks prior to admission to hospital, dropsy of the ankles had come on. He had been continuing to work two days a week till within a short time of coming to hospital, in spite of his breathlessness being so bad that in walking to and from work it was his custom to rest at each lamp-post as he came to it in order to get his breath. Also when digging and doing similar hard work, he would have to stop and wait to get his breath every few minutes. When he found that his breathlessness did not admit of his walking as far as from one lamp to the next, he thought it was time for him to come to the hospital, and he was admitted. When first seen, there was

Myocardial Weakness mainly involving the Right Ventricle

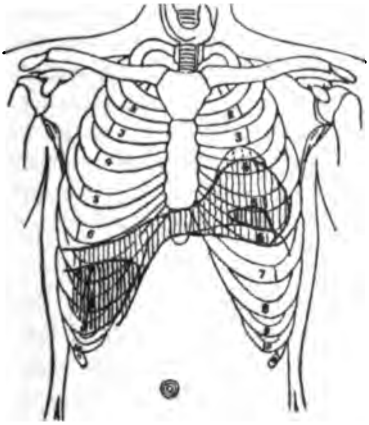


Fig. 20.

H. H., aged sixty. Weak right ventricle; stronger left one.

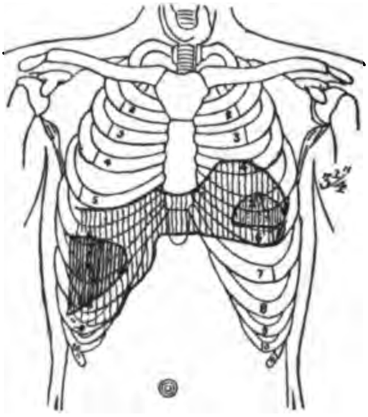


Fig. 21.

H. H., aged sixty, June 14, 1911. Heart stronger; cardiac area larger.

well-marked cyanosis and some dropsy of the legs (of which the kidney disease may have been a contributory cause). There was no abnormal distension and very slight pulsation of the veins of the neck, and although they were moderately full the supraclavicular fossæ were distinctly hollow.

There was no dilatation of the heart to the right, and the cardiac dullness was somewhat diminished in area. There was no absolute dullness except a little in the fifth interspace, and the relative dullness barely reached to the left edge of the sternum. The absolute liver dullness was small and did not reach to the costal arch.

There was no cardiac impulse to be felt, and the apex was probably in the fifth interspace about midway between the nipple line and the anterior axillary line. The heart sounds were short and wanting in tone over both right and left ventricle, and no murmurs were present. The diagram shown in fig. 20 was taken about a fortnight after admission when his cyanosis and dropsy had gone, and his dyspnœa was much less; but it shows the heart in much the same condition as on admission.

Here we have marked cardiac failure—as judged by his great dyspnœa on exertion associated with no abnormal fullness of the veins, a small liver, and the right side of the heart if anything smaller than normal. There was a considerable amount of emphysema; for there was very little, if any, true expansion of the thorax, and the upper edge of the liver was a little low, and breathlessness, if due to this cause, ought to have been associated with an over-full venous system and a labouring right heart with loud sounds and a distended liver.

Here we have clear evidence of muscular weakness of the right ventricle, and, as a further comment upon what has recently been said, it might be added that the evidence was against there being any very marked rigidity of the heart wall, for the following reasons: The ventricle was smaller than normal, which suggested the probability that it had diminished in size somewhat, proportionally to the lessened amount of blood it was able to expel, and also there was some variation from time to time in the size of the cardiac area, as shown in the second diagram, which was taken when the heart was a good deal stronger, and shows some increase upwards of the cardiac area as compared with the former one.

And now as regards the left ventricle. It is evident that the left side was fairly strong, otherwise he could not have continued working for many weeks—if not months—in spite of very considerable dyspnœa. This statement is also supported by the condition of the pulse, which was hard and wiry; and on testing with the sphygmomanometer was found to withstand a pressure of 280 mm. of mercury. The arterial wall was not specially hard, and the pulse did not give to the finger the impression of its being a high tension pulse.

It was described as being a pulse of very small volume, moderately well sustained, and subsiding under the finger between the beats about as rapidly as normal.

The nutrition of the left ventricle must have been fairly good for it to maintain such a blood pressure. The smallness of the volume was doubtless due to the deficiency in the amount of blood which the right ventricle was able to keep in circulation, and in this connection the questions arise, How far was the high blood pressure due to some pathological change in the condition of the arterioles, the result of the kidney disease? and, How far was it a compensatory phenomena necessitated by the small volume of blood which was available for the left ventricle?

The fact that the apex was about one inch outside the nipple line and in the fifth space might be taken as suggesting that there had been in the past dilatation due to the overaction necessitated by arterial contraction such as results from kidney disease.

The presence of some dilatation of the left ventricle supports the evidence given by the right ventricle as to the absence of any marked degree of rigidity of the heart wall. It is probable that the possession of an unusual degree of distensibility accounted for the unusual features of this case. For it is not common to find a man able to continue working, as he did, in spite of such a great amount of breathlessness. It is, I think, fair to assume that if the right ventricle had been more rigid than it was, it would not have been able to adapt itself to the altered conditions of the circulation, and that, under such circumstances, the man's condition would have been much less satisfactory than it actually was.

And now we had better conclude this essay by summarising the various points which are apt to give rise to difficulty in the diagnosis of heart failure in later life.

In the first place, we must mention the main cause—namely, that the ordinary and easily recognised form of failure with enlargement of the heart does not constitute anything like so large a proportion of all cases as it does earlier in life. In other words, we more often meet in later life cases where failure is unaccompanied by enlargement and evidence of venous plethora, and therefore we have more cases where the signs of failure are few and require careful observation to recognise them.

Again, another set of difficulties arises from the tendency of the heart to partake in the general increase in stiffness (or rigidity) which is apt to characterise other tissues of the body as age advances. This also tends to lessen the likelihood of obvious signs of heart failure being found, whether they be of the nature of enlargement or of diminution in its size as clinically recognised.

Another set of difficulties arises owing to the changes that are apt to take place in the arteries in later life and the frequency with which high blood pressure and arterial disease occurs, rendering the pulse, very often, quite an uncertain guide, and because an apparently strong pulse may really be a dangerously weak high-pressure one.

Arterial disease is also accountable for another set of diagnostic difficulties that we have to face—namely, those due to the fact that in later life a common cause of heart failure is the gradual interference with its nutrition, which disease of the coronary arteries induces, and the fact that such interference is usually greater on one side of the heart than the other. We therefore have to be prepared to meet with cases whose one ventricle is strong and the other one is weak.

This same onset of malnutrition in later life may be difficult to recognise in hearts that are already enlarged as the result of valvular disease or of overstrain occurring earlier in life.

Here, again, the 'negative' signs (using the word in its mathematical sense) of myocardial failure interacting upon the positive ones of the pre-existing heart failure will not give very easily recognised evidence of their presence.

A very large number of difficulties, in such cases as we are speaking of, depend upon the fact that a weak heart circulates less blood than normal, and that, as a result, the venous circulation is lessened in volume, in contrast to failure in a strong heart, where the volume of blood in the veins—and especially the thoracic ones—is increased.

We may therefore meet with a large number of apparently contradictory evidences of heart failure and physical signs—such as increased fullness of the veins, diminution in the size of the right ventricle, &c.—which may show either improvement or deterioration in the condition of the heart, according to the circumstances under which they occur. These paradoxes appear worse on paper than they prove at the bedside; but it is necessary to be on the alert for their occurrence. Here is a list of some of them :—

Increased fullness of the veins in a moderately weak heart means deterioration, and in a very weak one means improvement; and, conversely, lessened fullness means improvement in a heart only moderately weak, and deterioration in a very weak one.

Extreme emptiness of the veins of the neck is a very good sign in a moderately weak heart, and a dangerously bad sign in a very weak one.

As regards the heart: dilatation of the right or left ventricle in a moderately weak heart is the commonest sign of heart failure, but in a case of severe myocardial weakness dilatation is the usual sign of returning strength. Again, the lessening in the size of a slightly dilated ventricle in a moderately weak heart would be a sign of improvement, but in a very weak one it would probably mean dangerous muscular failure.

Absence of dilatation in pneumonia would be a good sign if the heart were strong; but it may also (*vide* p. 97) be a sign of very dangerous myocardial weakness.

The same rule holds with regard to the heart sounds; for, where they are unduly loud—say, those of the right ventricle when the left is failing—a lessening in their loudness may be due to the recovery of the left side, or to the onset of muscular weakness on the right side, as the result of the overstrain—a very alarming condition.

There are also some points with regard to the liver; for, while a diminution in the size of a liver which is enlarged by tricuspid regurgitation is usually a good sign, a sudden lessening of its size might be a sign of the onset of myocardial weakness, as in the case mentioned at p. 63.

Also in this connection the fact must be remembered that a tense liver may give a resonant note by conduction of vibrations to and from an air containing viscus below it, whereas when less distended with blood it does not do so. Therefore, as a case of tricuspid regurgitation with an enlarged liver improves, the area of absolute liver dullness in the epigastrium may sometimes be found to increase downwards, and the incautious observer may fear that the patient is getting worse. This mistake can easily be prevented by noting the level at which the liver edge can be felt, it will be found in such a case to rise as the level of the absolute dullness descends.

ESSAY II.—ON THE VALUE OF AN ABNORMAL RISE IN THE AVERAGE LEVEL OF THE DIAPHRAGM AS A GUIDE TO THE VOLUME OF BLOOD IN ACTIVE CIRCULATION[1]

THE object of this essay is to bring forward clinical evidence in favour of the following propositions :—

1. When, from any cause, the total volume of the blood in circulation is materially diminished, the total bulk of the intrathoracic viscera is correspondingly diminished by the relative emptiness of the thoracic blood-vessels, especially those of the lungs. This diminution in the bulk of the intrathoracic contents shows itself by an elevation of the diaphragm, which has to be maintained at a higher average level than the normal in order to adjust the cubic content of the thorax to the altered volume of its contained viscera.

2. That such a rise of the diaphragm may frequently be detected clinically, and, when present, is of value in diagnosis and affords important indications for treatment.

NORMAL POSITION OF THE DIAPHRAGM

The position of the diaphragm can be recognised clinically by ascertaining the upper level of the gastric resonance in the left nipple line, or of the upper border of the liver in the right nipple line. In both cases percussion is best done with the patient recumbent; and in the case of the gastric resonance the recumbent posture is essential to accuracy, because in the erect posture the gaseous contents of the stomach, if scanty, may rise up into the arch of the diaphragm, and the dullness of the liquid contents of the stomach be continuous with the dullness of the left lobe of the liver and the heart.

---

[1] Read before the Birmingham Branch of the British Medical Association. See the *British Medical Journal*, April 27, 1907.

Of the two methods named, the percussion of the gastric resonance is easier and more reliable for the following reasons:—

1. There is much variation in the upper level of the liver dullness of healthy individuals, dependent upon the degree to which the lungs have been expanded by exercise or athletics, &c.

2. The respiratory movements of the lower edge of the lung render exact accuracy of record as to its level impossible.

3. The level of the diaphragm on the right side is more apt to be interfered with by ill-defined pathological conditions of the lung than is the case with the level of the central tendon which underlies the heart.

Gastric Resonance

For the study of the gastric resonance it is necessary, as before mentioned, for the subject to be lying on his back, so that any gas in the stomach may come to the front and give a contrast with the dullness of the left lobe of the liver and the heart, which lie above it.

The determination of the area over which the gastric resonance is obtainable is sometimes rendered more easy by listening to the area over which 'reverberation' can be heard when some continuous sound is communicated to the air in the stomach. This is simply done by means of a short rough rod, which is pressed over the stomach area and scratched regularly with the fingernail. The echo, similar to the bell sound in pneumothorax, is clearly heard over the whole gastric area, but is sharply limited to it (*vide* Essay XVI, p. 480).

As a rule the results obtained by percussion and by noting the area of gastric reverberation coincide very closely with each other.

By these methods the normal level of the upper limit of the gastric resonance is found to be in the sixth left interspace. It sometimes in health reaches the upper border of the sixth rib, but, as a rule, when it reaches the fifth interspace it must be considered pathological. Only once or twice have I found well-marked gastric resonance in the fifth interspace in a person who was apparently in perfect health, and under circumstances which pointed to its being the normal level for the individual concerned.

UPPER LEVEL OF THE LIVER DULLNESS

Although the percussion of the upper border of the liver is of less value than that of the stomach, where exact observations are desired upon the level of the diaphragm, it nevertheless gives us important information as to the condition of the diaphragm, for its elevation above the normal level may be very great when the diaphragm is high. Thus in one patient (Case XI) there was distinct relative liver dullness up to the middle of the third rib in the right nipple line.

The percussion of the upper border of the liver dullness is also of considerable value, because it often gives an indication as to the amount of arching of the diaphragm present, if the distance between the level of the absolute dullness and that of the relative liver dullness be carefully observed, and the changes during respiration noted. Thus a wide area of relative dullness suggests an abnormally arched diaphragm and a sudden change from full lung resonance to absolute liver dullness suggests a flatter diaphragm, as in emphysema.

This abnormal wideness of the area of relative dullness, indicating a highly arched diaphragm, is well shown in some of the following diagrams—namely, figs. 10, 19, 27, 31, 41.

OBSERVATION OF THE LOWER EDGE OF THE LIVER

The careful mapping out of the lower edge of the liver dullness is also of importance in the study of the level of the diaphragm, because as the diaphragm rises, the rise of the lower border of the liver dullness is often more marked than the upper. It is not uncommon to find in such cases a total disappearance of the absolute dullness of the liver, internal to the right nipple line, accompanied by a marked rise in the lower edge of the liver, sometimes to the extent of 2 inches or more above the costal arch. This change is well shown in figs. 26, 31, 38, 40. It seems as if the liver slips back into the hollow of the diaphragm, and its lower anterior edge is thus drawn up and back so much that it barely comes in contact with the ribs anteriorly at all; and the intestinal or gastric resonance meets the lung resonance over this area. This strange change in the shape of the absolute dullness of the liver will sometimes develop in a week or so, and then as speedily give way to the normal relationship of the liver dullness to the costal arch, when the conditions causing the ascent of the diaphragm subside (Case IV). (See also pp. 187, 188.)

SCOPE OF SUBJECT

Though this essay does not deal with cases in which the level of the diaphragm is altered by some obvious physical cause—such as the pressure of distended abdominal viscera, or the traction of a collapsed or fibrosed lung—nevertheless, it may be well to say a few words as to the changes which take place when the left lung shrinks from healing tuberculosis, or remains unexpanded after compression by extensive pleurisy. In these cases we know that the diaphragm is drawn up by the lessened size of the left lung, and as the changes which take place in the level of the gastric resonance are precisely those which occur in the cases dealt with in this paper this may be taken as evidence in favour of the points raised.

CASES SHOWING ELEVATION OF THE DIAPHRAGM FROM COLLAPSE OF THE LEFT LUNG

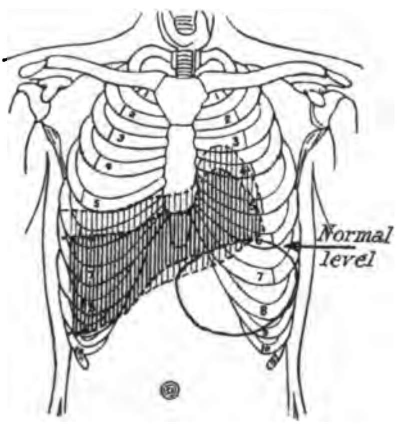


FIG. 22.—CASE I: NORMAL CHEST.

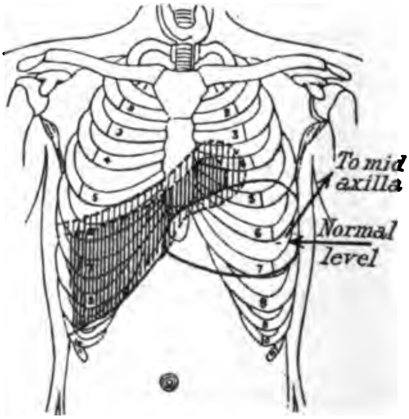


FIG. 23.—CASE II: COLLAPSE OF LUNG.

Diaphragm high on left side only.

I give the notes and figures of two such cases, one moderate and one extreme.

CASE II.—MODERATE ELEVATION

The heart is drawn up a little to the left by the rise of the diaphragm, but there is a marked rise in the gastric resonance (*vide* fig. 23) not only in the left nipple line, but much more so in the axilla. Instead of there being no gastric resonance in the axilla, as in the normal chest, in this case the resonance reached the fourth interspace in the nipple line and extended nearly to the anterior axillary line, 4 inches from the sternum. In the fifth space it reached 5½ inches, and in the sixth space there was full gastric resonance and reverberation to the mid-axillary line, 6½ inches from the sternum; the lower border was at the eighth rib.

This condition developed on the rapid absorption of pleuritic effusion, and in eleven days the upper border of the gastric resonance had returned to the normal level, and there was none in, or external to, the anterior axillary line.

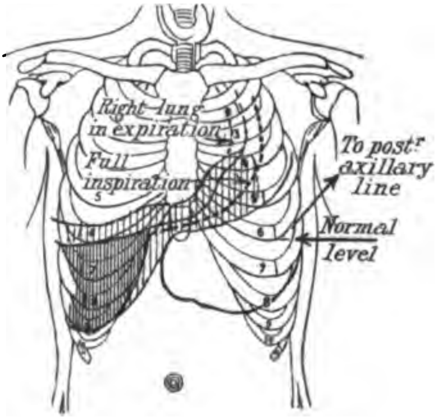


FIG. 24.—CASE III: COLLAPSE OF LUNG.

Very high left diaphragm.

CASE III.—EXTREME ELEVATION

There was complete non-expansion of the left lung (fig. 24). The right lung crossed over, and its left border could be detected just internal to the left nipple line (2¼ inches from the sternal edge) on expiration, and during full inspiration it crossed to beyond the nipple line (more than 4 inches from the left edge of the sternum) in the second, third, and fourth interspaces. This shows the extreme shrinkage of the left lung. The diaphragm also was correspondingly high, as judged by the level of the gastric resonance.

The expansion of the right lung pushing down the heart prevented any great rise of the diaphragm in the nipple line, and it was only at the fifth interspace; but in the axilla there was full gastric resonance to the posterior axillary line in the sixth space, 8 inches from the middle line. Its lower border was in the eighth space.

HIGH DIAPHRAGM IN ENTERIC FEVER

Although, in common with all those who carefully percuss the cardiac dullness, I have long been conscious that there is occasionally abnormal resonance below the cardiac area, I did not give the subject special attention till the following case brought it forcibly under my notice :—

A man suffering from an extremely severe attack of enteric fever presented very great asthenia, but no complications of any sort until the end of the third week. He then became decidedly feebler, although the temperature was not specially high, and the condition of the abdomen was satisfactory. He had had no diarrhœa. This asthenia was suggestive of the virulence of the poison he was absorbing; but as his condition became worse it was evident that some other cause was at work. The abdomen from being moderately full became abnormally retracted, and his face and limbs gave the appearance of extreme emaciation. This rapid change in his appearance was evidently due to lack of liquids in the body. He was taking three pints of milk daily, besides some water. I tried the effect of a subcutaneous saline injection of half a pint or more; it was rapidly absorbed, but as rapidly excreted by the kidneys, and the patient's condition was in no way improved. There was a peculiar expiratory type of dyspnœa, which I have now learned to associate with this condition, and also a decided elevation of the diaphragm, the gastric resonance reaching well into the fifth space, and the liver being decidedly above its usual level in the right nipple line. He died within two or three days of these symptoms becoming noticeable.

Post-mortem, the diaphragm was at the third rib on the left and at the fourth rib on the right side; the heart was small, its lower border at the fourth interspace, and all the organs dry and bloodless.

In thinking over the cause of death, I was forced to the conclusion that death was practically due to starvation, owing to the interference with intestinal absorption, which resulted from inflammatory changes in the mesenteric lymphatics, and my explanation of the case was as follows :—

This defective absorption from the intestines, coupled with the tissue-waste due to the fever, would presumably in course of time lessen the proteids in the body to such a degree that the total volume of the blood would have to diminish in order to prevent it from becoming too dilute. This diminution at last became so extreme that the proper nutrition of brain and heart was no longer possible, and the patient died. The explanation of the elevation of the diaphragm which appeared to be most probable was that the diminution in volume of the circulating blood caused such a lessening in the size of the heart and such emptiness of the pulmonary artery and great vessels that the volume of the heart and lungs was so materially diminished that the diaphragm had to rise so as to diminish the cubic content of the thorax to a corresponding extent.

I have had some confirmation of this explanation of the preceding case, for I have on several occasions seen the same symptoms appearing in severe cases of enteric fever. When I have detected the peculiar sighing breathing and the elevation of the diaphragm, associated with signs of lack of fluid in the body, I have been able at once to remove the dangerous symptoms by the administration of peptones and of milk sugar,[1] the absorption of which takes place through the portal vein and not through the intestinal lymphatics.

It is now some years since I have seen such a case, because I am careful to guard against such malnutrition.

HIGH DIAPHRAGM IN LIMITATION OF FOOD

If the above suggestion were accurate and a limitation of the intake of nutritive material would (if carried to excess) cause a diminution in the total volume of the blood, and show itself by a shrinking of the thoracic contents, the same phenomenon of high diaphragm ought to be found where semi-starvation is adopted as a therapeutic measure—for instance, in the treatment of gastric ulcer.

For many years I have noticed that as a rule in cases of gastric ulcer there is a marked rise in the diaphragm after some

---

[1] The administration of sugar in enteric fever must only be resorted to in such cases as these. In ordinary cases it appears greatly to increase the severity of the symptoms—presumably, by feeding the bacilli more than it feeds the patient.

CASE OF GASTRIC ULCER, SHOWING A HIGH DIAPHRAGM DUE TO STARVATION

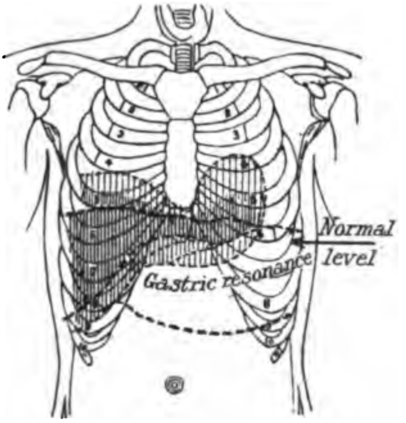


FIG. 25.—CASE IV: GASTRIC ULCER.

March 7. Before limitation of food.

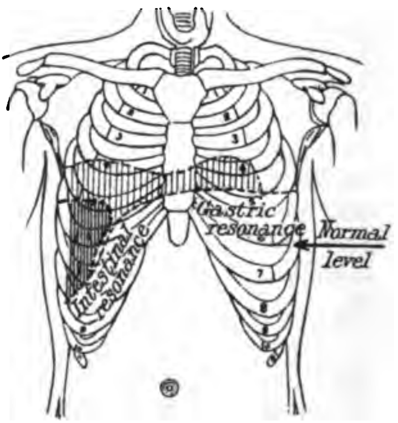


FIG. 26.—CASE IV.

March 13. After six days of rectal feeding.

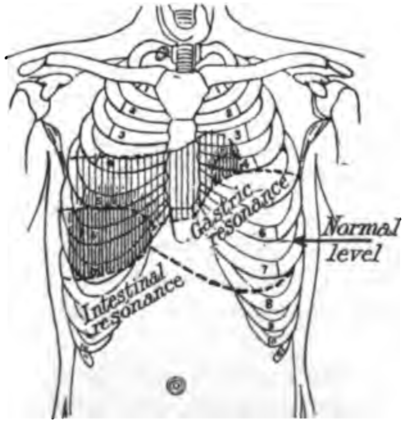


FIG. 27.—CASE IV.

March 15. More food given; heart larger.

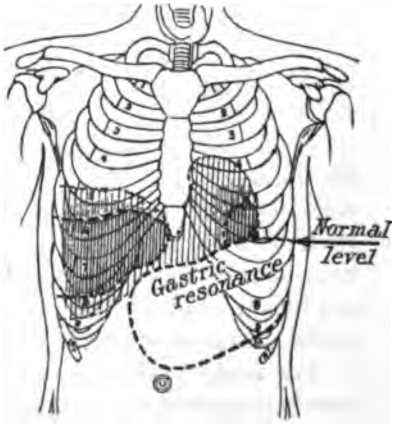


FIG. 28.—CASE IV.

March 19. Food increased.

days of strict limitation of food, as where the patient is fed only by nutrient enemata.

The amount and the duration of the rise varies much in different cases. In some the gastric resonance will, after a a few days of rectal feeding, rise into the fifth interspace, or even into the fourth, and as rapidly return to the normal level on the resumption of full feeding. In others the rise may take place more slowly, attaining its maximum height after the cessation of rectal feeding, and only returning to the normal level after the patient has been on ordinary diet for some time.

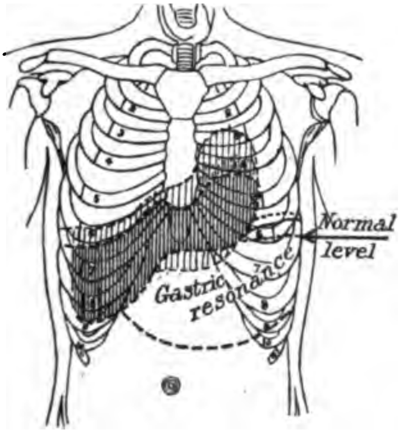


FIG. 29.—CASE IV.
March 29. Transient cardiac dilatation.

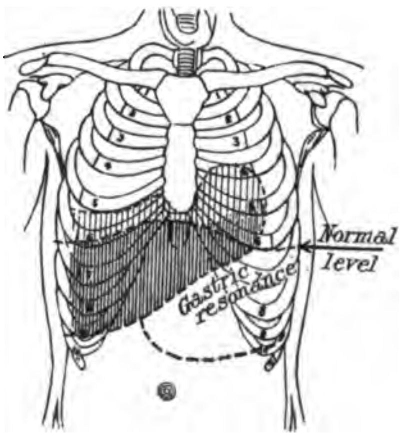


FIG. 30.—CASE IV.
March 30. Practically normal.

The following case shows an unusually rapid rise and fall of the diaphragm, and is illustrated by figs. 25–30 and by a table showing the details as to the rise and fall of the diaphragm.

CASES OF GASTRIC ULCER

CASE IV.—GASTRIC ULCER

Kate G., aged thirty-eight, admitted to hospital March 6, 1906 (figs. 25–30). There was a history of dyspepsia of many years' standing, and an attack of hæmatemesis six years ago. Present illness dates back to an attack of influenza three weeks ago; ten days ago, an attack of vomiting was followed four days later by hæmatemesis, half a pint of blood being brought up.

DETAILS AS TO THE RISE AND FALL OF THE DIAPHRAGM IN CASE IV

Date	Food	Stomach level	Upper border of liver dullness			Lower border of liver	
			Absolute	Relative	Width of relative dullness	Situation	Distance from costal arch
March 7	Fed entirely by the rectum	6th rib (U.B.)	5th rib (L.B.)	4th rib (L.B.)	1¼ in.	—	1½ in.
„ 13		5th rib (U.B.)	5th rib (Mid.)	4th rib (Mid.)	—	—	2 in.
„ 14		4th rib (L.B.)	—	—	—	—	—
„ 15	1 oz. per hour by mouth	4th rib (L.B.)	5th rib (L.B.)	4th rib (U.B.)	2 in.	7th rib (L.B.)	2½ to 3 in.
„ 18	Low diet; enemata stopped	6th rib (L.B.)	5th space	5th rib (L.B.)	1¾ in.	8th rib (Mid.)	1¼ in.
„ 19							
„ 23	—	6th rib (L.B.)	6th space	5th rib (Mid.)	¾ in.	9th rib.	At C.A.
„ 26	Fish diet	—	—	—	—	—	—
„ 30	—	6th rib (L.B.)	6th rib (Mid.)	5th rib (L.B.)	—	9th rib (L.B.)	Below C.A.

Explanation.—U.B., upper border. L.B., lower border. Mid., middle. C.A., costal arch.

On admission, much pallor. Pain after food, and epigastric tenderness were noted. Examination of the abdomen showed that the diaphragm was rather high, the gastric resonance reaching up to the upper border of the sixth rib, and the lower border of the liver being 1½ inches above the costal arch. There was 1½ inches of relative liver dullness in the right nipple line. She was fed entirely by the rectum for a week, and then the food was gradually increased.

The rise of the diaphragm, and its return to normal, are well shown in figs. 25, 26, 27, 28, 29, and 30, and also in the table given. With regard to fig. 29, the transient dilatation upwards of the heart is of the type usually seen in anæmia.

The following is such a case:—

CASE V.—GASTRIC ULCER WITH SEVERE LOSS OF BLOOD

Nelly Y., aged twenty, admitted January 18, was kept on nutrient enemata only till January 20, then allowed one pint of milk a day as well. This diet was increased gradually till by February 10 she was taking a full fish diet.

The stomach resonance and the liver dullness (figs. 31–33) continued to rise till January 29, when they stood at the upper border of the fifth left and fourth right ribs respectively. At this date she had been for two days on an ordinary bread-and-milk diet. By February 10 there was still some elevation of the diaphragm, but the relationships of the organs were more normal.

HIGH DIAPHRAGM AFTER SEVERE HÆMORRHAGE

Closely allied to these cases are those where, after a sudden severe hæmorrhage, the diaphragm may be found to be an interspace or more above the normal level.

High diaphragm, if looked for, can very often be found in such cases as are blanched by severe hæmorrhage, and the gradual return of the diaphragm to the normal level can be observed as the patient recovers (fig. 34).

CASE VI.—HÆMATEMESIS

Louisa C., aged thirty-three, admitted with very severe hæmatemesis. She was so blanched that the foot of her bed had to be kept elevated to prevent faintness.

A CASE OF GASTRIC ULCER SHOWING A HIGH DIAPHRAGM

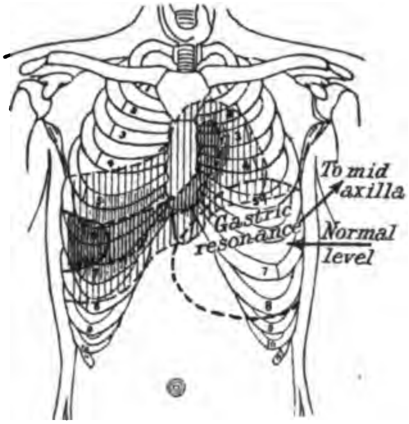


FIG. 31.—CASE V: GASTRIC ULCER.

January 20. After three days of rectal feeding.

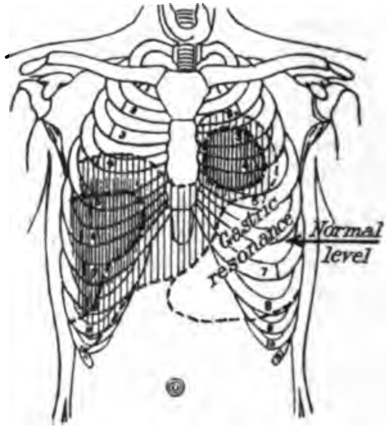


FIG. 32.—CASE V.

January 29. More food; diaphragm lower.

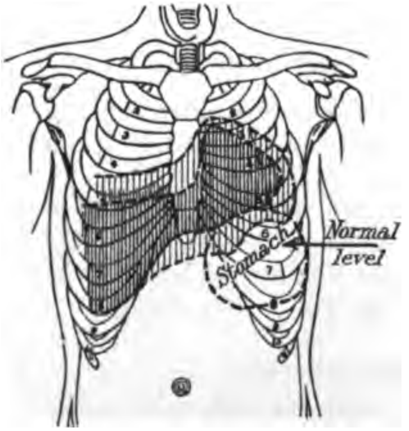


FIG. 33.—CASE V.

Diaphragm lower.

HIGH DIAPHRAGM THE RESULT OF HÆMORRHAGE

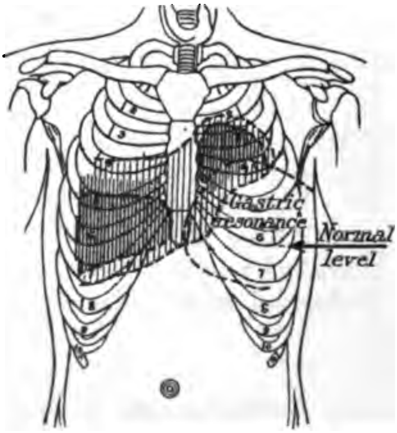


FIG. 34.—CASE VI: HÆMATEMESIS.

HIGH DIAPHRAGM IN ANÆMIA

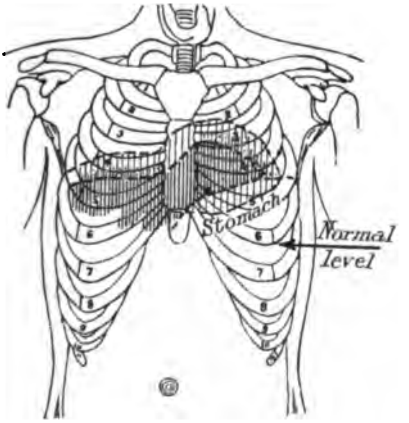


FIG. 35.—CASE VII: ANÆMIA.

High diaphragm and dilated heart.

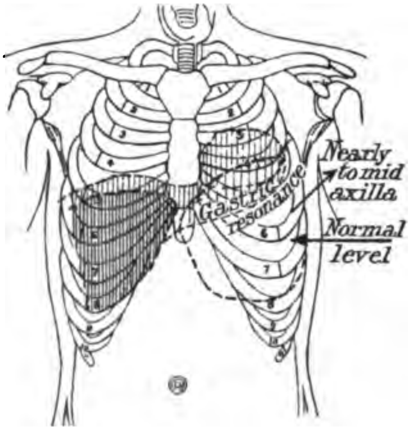


FIG. 36.—CASE VIII: ANÆMIA.

February 15. Diaphragm very high in axilla.

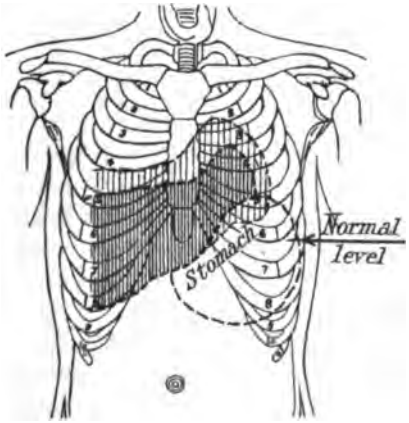


FIG. 37.—CASE VIII.

March 29. Nearly normal.

HIGH DIAPHRAGM IN MYXŒDEMA

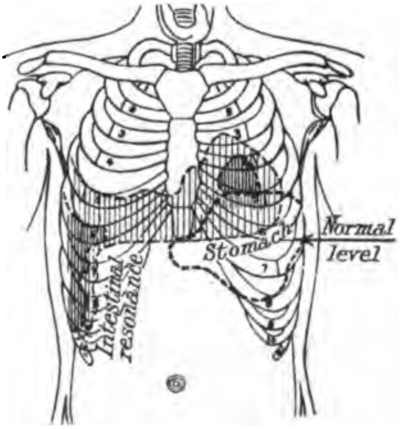


FIG. 38.—CASE IX.

Neurasthenia.

The figure (34) shows the marked rise in the level of the gastric resonance, and of the lower edge of the liver; also the broadening of the relative liver dullness. In this case there was full intestinal resonance for 2 inches above the costal arch in the right nipple line.

HIGH DIAPHRAGM IN ANÆMIA

In anæmia, from causes other than loss of blood, the same phenomenon of high diaphragm, though by no means constant, is very frequently noticed. In a series of consecutive observations upon cases of anæmia and chlorosis I found a well-marked rise of the diaphragm—that is, to the fifth interspace, or higher—in about one-third of the cases; but I was not able to determine the exact reason for the difference in this respect without a fuller investigation into the blood changes present in each case than the time at my disposal permitted.

In true chlorosis it has been stated that the volume of the blood is, if anything, increased; and if this be proved to be so, the observation of the level of the diaphragm may prove of diagnostic value in these cases.

The reason for the rise of the diaphragm in anæmia cannot be definitely stated. It may be the result of deficiency of proteids, or it may be due to deficiency of iron or some other necessary constituent of the blood, acting in the same way as the deficiency of food acts.

This hypothesis may be expressed in general terms by saying that the total volume of the blood diminishes because there is a deficiency of some one or more of the essential constituents of the blood, and therefore the total volume of the blood in circulation must be lessened in order to keep the percentage of that substance at its irreducible minimum.

In these cases of anæmia the area of cardiac dullness is, as a rule, larger than in the cases previously mentioned. This is due to the frequent presence of dilatation of the right ventricle.

In connection with these changes in anæmia there is one result of high diaphragm which may have been misinterpreted in the past—namely, the displacement upwards of the apex beat. In papers I have written and seen upon this subject this upward and outward displacement of the apex of the heart in anæmia has been explained solely by changes in the heart itself. I am now sure that in some of the cases, at all events, the rise in the diaphragm must play some part in the upward displacement.

CASE VII.—SEVERE ANÆMIA.

Cissy G., aged thirty-one. The gastric resonance reached well into the fourth left interspace, and in the fifth interspace there was well-marked gastric resonance throughout the whole interspace from the sternum to the anterior axillary line (fig. 35). The heart was displaced upwards, and, owing to the anæmic dilatation of the right ventricle, there was a larger area of dullness than in the cases of gastric ulcer without marked anæmia. There was some cardiac impulse in the fourth interspace, but the maximum impulse was in the third left interspace external to the nipple line (3 inches from the sternum). In the second left interspace there was relative dullness and pulsation for 2 inches from the sternum.

CASE VIII.—ANÆMIA

Ada F., aged seventeen. Admitted to hospital February 15, with anæmia and severe chilblains. On admission the area of cardiac dullness was small, there being no absolute dullness, and the gastric resonance was very high—namely, up to the middle of the fourth rib in the left nipple line. There was also a great increase towards the axilla, for gastric resonance and reverberation were clearly obtainable in the fourth interspace near to the mid-axillary line (fig. 36). In the course of ten days the diaphragm was lower, the gastric resonance reaching only to the fifth rib, and only extending into the axilla in the sixth and seventh interspaces. After being in hospital six weeks the stomach, though still up to the fifth rib, did not reach into the axilla at all, and the cardiac dullness was larger, showing some anæmic dilatation.

HIGH DIAPHRAGM IN NEURASTHENIA

In many other diseases a high diaphragm may occasionally be met with ; but in cases of neurasthenia it is of such relatively frequent occurrence as to raise the question whether in this condition some of the chemical or organic constituents of the blood are not deficient in amount.

CASE IX.—NEURASTHENIA

A case of a somewhat similar nature is that of a labouring man who was sent to me as a possible malingerer, because he looked comparatively hale and hearty, but nevertheless complained of great lassitude and inability to work. His colour was good; there was no complaint except of a little atonic dyspepsia. Physical examination showed nothing except a high diaphragm, the gastric resonance reaching to the fourth left interspace and the absolute dullness of the liver being absent internal to the right nipple line (see fig. 38). There were some symptoms which suggested the possibility of deficiency of thyroid secretion, and treatment by thyroid extract entirely cured him.

COMMENTARY

We must now discuss the explanation of the phenomena described and illustrated by the diagrams given.

Is it right to conclude that elevation of the gastric resonance on the left side and of the liver dullness on the right side implies certainly an elevation of the diaphragm? As the phenomena described, though less marked, are still present in the erect as well as in the recumbent attitude, and as the rise in the gastric resonance can usually be noticed far back in the axilla as well as in front, an affirmative answer must be given. The diaphragm is high in such cases.

Granted, then, that the diaphragm is high in the cases of which we have been speaking, what is the explanation of its rise in this marked manner?

It must either be drawn up by suction from above, or pushed up by pressure from below; for we know of no power inherent in itself whereby the diaphragm could rise and compress the thoracic viscera.

But of these two forces it is easy to eliminate pressure from below as a cause of the elevation of the diaphragm. In all these cases the abdomen is empty or normal in appearance, and there is certainly no increase of intra-abdominal pressure.

Then, again, it cannot be due to some peculiar action of the stomach, because the rise is bilateral, involving the liver as well as the stomach. Moreover, a glance at the diagrams will show that the lower level of the stomach is often raised as well as the upper border, and I have never in these cases noticed any prominence of the stomach in the epigastrium, such as is always noticeable when the stomach is sufficiently distended and tense to exert pressure on the thoracic viscera.

Moreover, the pitch of the reverberation note as heard over the stomach is not of the high and varying character noticeable where the organ is distended and undergoing contraction.

We must therefore conclude that the diaphragm is drawn up as the result of some change in the intrathoracic pressure.

What intrathoracic force can do this ?

Now, although there is a danger of error in reasoning when a process of exclusion is used, we may, I think, safely resort to it in this instance.

The thorax contains practically only four things whose variation could materially alter the volume of the thoracic contents: the lung tissue, including the bronchi; the air in the lungs and bronchi; the heart and blood-vessels; and the blood.

Now, some of these variable factors are easily excluded. (1) There can be no abnormal suction exerted by the air in the lungs, as there is no mechanical interference with the entrance or exit of the air from the thorax. (2) The actual bulk of the lung tissue is not changed; the lungs in the cases spoken of are healthy.

The agency causing elevation of the diaphragm must, therefore, be either a variation in the cardiac and vascular tissue, or in the degree of distension of the heart and blood-vessels in the thorax (including those of the lungs) with blood. As the cardiac tissue is practically constant in bulk from day to day, the factor we are searching for must consist of a lessening in the amount of blood contained in the lungs, heart, and great vessels in the thorax.

When we consider how large a proportion of the lung tissue consists of capillary vessels, how much blood the veins of the thorax and the heart itself are capable of holding, it is clear that a decided diminution in the volume of blood circulating in the thorax might cause a distinct lessening in the thoracic contents, and the space thus left vacant would have to be filled up either by air or by a corresponding lessening of the cubic content of the thorax by means of a rise in the level of the diaphragm. There is, therefore, physiological justification for the theory that the rise in the diaphragm is caused by a lessening in the amount of blood in the thorax.

These theoretical considerations are in accord with the facts of the cases mentioned, for in them there were conditions leading to malnutrition of the body, and presumably also lessened power of regenerating the blood.

One theoretical consideration which follows upon the clinicial facts here brought forward (if the explanation given be correct) is that there must be a limit below which the specific gravity of the blood (or the percentage composition of its various constituents) is not allowed to fall. Where there is a lessened intake of food, when the impoverishment of the blood reaches this minimum limit, it can go no further without damage to the organism; and consequently if the lessened intake of food continues the total volume of the blood must be lessened, in order that the minimum limit be not passed.

It is possible that this law of an irreducible minimum may hold for many of the constituents of the blood, and that the high diaphragm of neurasthenia is due to the deficiency in the blood of some chemical substance, or some internal secretion. The case I have already referred to of the labourer who suffered from neurasthenic symptoms, and had a high diaphragm, and was cured by the administration of thyroid gland, seems to point towards such a possibility.

High Diaphragm from Heart Weakness

Further evidence can, however, be adduced in favour of this proposition that a lessening of the amount of blood in circulation through the lungs and heart causes a recognisable diminution in their total bulk, and is followed by a rise in the diaphragm.

The volume of the blood circulating through the lungs is not dependent solely upon the total volume of the blood in the body; it is also dependent upon the strength of the heart.

A decided weakening of the heart as a whole might reasonably be expected to diminish the volume of blood circulating through the lungs. If a diminution of the total volume of the blood in circulation causes such emptiness of the lungs and vessels that the diaphragm has to rise so as to adjust the size of the thorax to the diminished bulk of its contents, then this same phenomenon would also occur if a weakening of the heart muscle results in an automatic diminution in the amount of blood in circulation in order to accommodate, as far as possible, the burden to the heart's power of bearing that burden. There is good clinical evidence that this takes place, for marked cardiac asthenia is extremely frequently accompanied by a rise in the level of the diaphragm.

This phenomenon of high diaphragm would not, however, be expected in cases in which cardiac weakness is accompanied by fullness of the veins—such as cases of failing compensation. In cases of valvular disease, nevertheless, the level of the diaphragm ought to be watched, as it may give very important indications.

Such a sudden weakening of the heart with a fall in the strength of the pulse is a well-known sign of the onset of pericarditis, and when this takes place in a young patient whose fibrous tissues have not yet become unyielding a well-marked rise in the level of the diaphragm will be found to be present.

This rise is especially noticeable where failure of the heart muscle from myocarditis or pericarditis occurs as a complication of rheumatic fever; for the rheumatic poison causes a softening of fibrous tissues, allowing the displacement of organs to occur more readily than if rheumatism were not present.

I give an illustration of an extreme case of this type.

The gastric resonance reached as high as the lower border of the fourth rib on the left, and the relative liver dullness reached the upper border of the third rib in the right nipple line, the lower edge of the liver dullness being at the lower edge of the sixth rib (Case XI, figs. 39 and 40). When the heart recovered, some eighteen days later, the gastric resonance and the liver dullness had both returned to the normal level. The details of the case are as follows :—

CASE XI.—RHEUMATIC PERICARDITIS

Emily E., aged fifteen, was admitted on October 29, very ill with rheumatism and pericarditis.

The diaphragm was very high, the gastric resonance reaching to the fourth rib, and the relative dullness of the liver was at the third rib in the right nipple line. The lower edge of the liver only reached to the lower border of the sixth rib. Three weeks later, on November 20, the condition had improved, but the diaphragm was still high, as shown in fig. 39. Within another three weeks the diaphragm was nearly back to its normal level (see fig. 40).

HIGH DIAPHRAGM IN PERICARDITIS

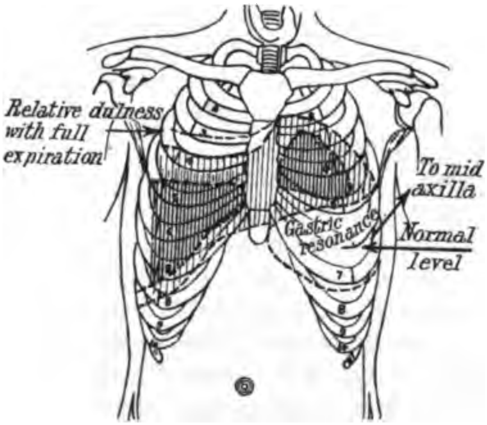


FIG. 39.—CASE XI: PERICARDITIS.
November 20. Very high right diaphragm.

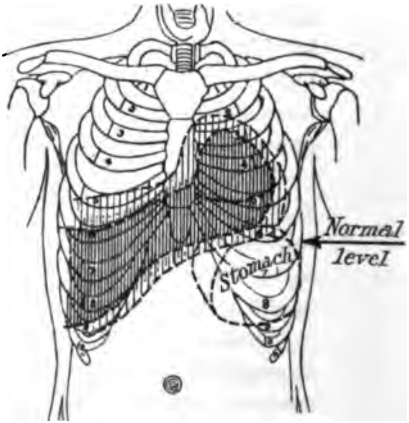


FIG. 40.—CASE XI.
December 8. Normal again.

There was very considerable dilatation of the right ventricle upwards to the left in this case.

A similar well-marked case was one in which sudden myocardial weakness occurred in a case of influenza complicated by acute colitis.

CASE X.—INFLUENZA

The heart weakness showed itself by a sudden fainting attack, during which the heart-rate fell to 40, and even to as low as 30 for a short time, if the pulse-rate were to be trusted. The cardiac asthenia was so extreme that for a fortnight or more the patient dare not raise his arms for fear of bringing on a fainting attack. During this time the diaphragm rose steadily. In spite of the fact that the patient was a young man, with voluminous lungs (due to his playing a wind instrument as a pastime), the gastric resonance on the fourth day of the illness reached into the fifth left intercostal space, and by the twelfth day reached the middle of the fifth rib in the left nipple line. Similarly, the absolute liver dullness by the twelfth day had risen from the sixth interspace to the lower border of the fifth rib. Accompanying this rise in the diaphragm there was a marked diminution in the cardiac dullness: in fact, it almost entirely disappeared, there being on the twelfth day of the illness no absolute dullness, and only a little relative dullness in the fourth space, internal to the nipple line but not reaching to the sternum.

HIGH DIAPHRAGM IN MYOCARDIAL WEAKNESS

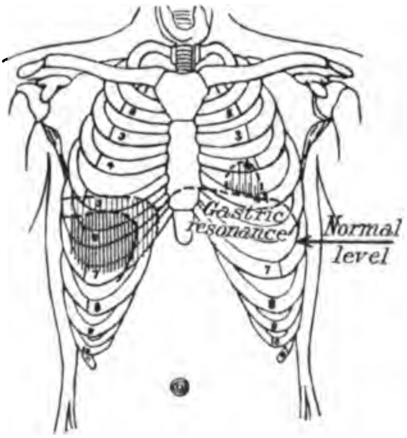


FIG. 41.—CASE X.
High diaphragm in sudden cardiac failure.

Here, then, we had marked cardiac asthenia associated with a rise in the diaphragm and diminution in the size of the cardiac dullness.

Within five weeks of this date the gastric resonance was back at the normal level, and there were 4 inches of cardiac dullness in the fourth interspace instead of under 2 inches, as on the date above mentioned, and the heart was much stronger. The patient made a perfect recovery, although appendicitis supervened on the acute colitis.

CLINICAL SIGNIFICANCE OF HIGH DIAPHRAGM

As regards the clinical significance of high diaphragm, the question has of necessity been dealt with in the above discussion of the phenomenon. It is an important danger signal in some cases of severe enteric fever. It is a useful guide as to the extent to which rectal feeding may be pushed. It must also be borne in mind because in cases of suspected rupture of a gastric ulcer the absence of the liver dullness cannot, as is now well recognised, be relied on as an indication of free gas in the peritoneal cavity.

In heart disease also the level of the diaphragm is a useful guide as to the condition of the circulation. If the weakness of the heart muscle be the prime factor, we shall expect a high diaphragm, unless, as in advanced age, the tissues be too rigid to yield easily ; if, on the other hand, some obstruction to the circulation—such as valvular defect—be the chief cause of failure, there will be excess of blood in the veins of the thorax, and a normal or a low diaphragm would be found.

Thus we have in the height of the diaphragm, as shown by the upper level of the gastric resonance, an indication for treatment and a help in the diagnosis and prognosis of disease, with which we can ill afford to dispense.

ESSAY III.—DIMINUTION IN THE AREA OF THE LIVER DULLNESS AS AN IMPORTANT SIGN OF MYOCARDIAL WEAKNESS OR OF DEFICIENCY IN THE TOTAL VOLUME OF THE BLOOD[1]

DIMINUTION OF THE LIVER DULLNESS DUE TO HEART WEAKNESS

THE subject of muscular failure of the heart is too apt to to be dealt with in our text-books of the present day in its more extreme and easily recognisable degrees, rather than in its earlier ones. Too little is said, and perhaps known, as to those signs which show that the tide has just turned in the wrong direction. The important thing in dealing with cardiac failure is to look for the straws which show the first change in the direction of the current, and not to wait until the current is evidently running strongly in the wrong direction.

If cardiac failure is not recognised till marked breathlessness, enlargement of the liver, and dropsy have commenced, most valuable time has been lost.

Among the recognised methods of ascertaining the working power of the heart there are very few which give us information as to the early commencement of pure muscular failure. In such cases reliance is mainly placed upon the patient's subjective symptoms; but yet, valuable though these be, some surer guide in the way of objective signs of muscular failure are highly desirable. Our recognised armamentarium is unfortunately very poor in such signs, in spite of the fact that they are by no means non-existent.

They are mainly to be looked for among phenomena due to deficient fullness of both the arterial and the venous system, in contradistinction to mechanical failure of the heart where emptiness of the arterial system is associated with fullness of the veins.

---

[1] Subject of a communication to the Dublin Meeting of the Association of Physicians, June 1909.

The clinical study of myocardial failure seems to prove that when the heart muscle is weak, the volume of blood in circulation is cut down so as to apportion the work to be done to the heart's power of doing it.

What happens to this blood, which is cut out of the circulation by the regulating mechanism of the body, I am not as yet prepared to say, as I have not yet been able to take up the investigation. In acute heart failure there can be no doubt that the blood is simply stored away in the abdominal veins or other reservoirs. In cases where the onset of heart weakness is more gradual and permanent, it is probable that the actual volume of blood in the body is diminished.

Also in these cases there is some interesting work to be done in answer to the question: Does the specific gravity of the blood and its corpuscular richness rise, owing to the elimination of water only from the blood, in a case of acute heart weakness where the condition persists, or is there a continuous storage without any concentration of the blood in active circulation, until the volume of the blood can be lessened by diminished activity of the blood-forming glands?

In slowly developing myocardial weakness the diminution will probably be brought about by diminished formation of blood.

Whatever the answer to these theoretical questions, the clinical fact remains that in heart weakness of pure muscular origin, apart from overstrain—absolute or relative—there is abundant evidence of diminution in the volume of the circulating blood.

The most valuable signs are those associated with emptiness of the vascular system, especially upon the venous side.

Of these the most important are a series of signs due to a diminution in the volume of the heart, lungs, and liver, when the blood passing through them is lessened in volume, smallness of the heart and liver being recognisable by percussion, and diminution in the size of the lungs and intrathoracic blood-vessels being measured by the altered position of the diaphragm, and by hollowing above the clavicles in cases where the diaphragm is not very elastic.

The hollowness above the clavicle and smallness of the veins in myocardial failure form a striking contrast to the fullness and pulsation seen in valvular disease, and other cases of mechanical failure, apart from weak heart muscle.

In addition to these, there are certain signs of emptiness of the arterial system—such as pallor, which is only present in special cases—and breathlessness, from weakness of the right ventricle. Also, not infrequently, cases are met with where the left ventricle is relatively weaker than the right, and in such a case it is not breathlessness which prevents the patient walking far or going up hills, but leg weariness—an aching pain in the calves, which gets worse the longer he walks, and which forces him to stop and rest—an 'intermittent claudication,' so-called, of pure myocardial origin.

By far the most valuable of these signs are those afforded by diminution in the size of the heart, the lungs, and the liver.

The diminished volume of the intrathoracic viscera, as measured by a rise in the position of the diaphragm was dealt with at pp. 168 *et seq.*, and it only remains for me to say here that enough stress is not laid in our text-books upon the diminution in the size of the heart, which may be detected by careful percussion. Enlargement of the heart is recognised as an important sign of altered heart's action, but diminution in its size is less apt to be recognised as a sign of muscular weakness than its importance deserves (*vide* figs. 42–52, and fig. 53).

In this paper, I shall deal with indications given by diminution in the size, real or apparent, of the liver as judged by careful percussion of its areas of dullness, absolute and relative.

It is only within the last two years or so that the fact of this diminution and its clinical importance were brought home to me, and my enlightenment came through the careful mapping out of the hepatic dullness, for the purpose of illustrating alterations in the level of the diaphragm. The diagrams were accurately drawn from nature, and doubtless many of those reading the paper on that subject (which is reprinted verbatim in the previous essay) felt, as I did, that the explanations given are not adequate to fully explain the appearances presented by many of them. They abundantly proved my thesis as to the rise of the diaphragm in cardiac weakness ; but the variations in the size of the liver dullness were greater than could be accounted for by the variations in the position of the diaphragm. It was on trying to understand my own diagrams that a truth came upon me which, to the best of my knowledge, has not as yet been recognised by the medical profession, but which ought, ever since the days when percussion was first used, to have been perfectly patent to every one. So blind are we, and so little able to leave the ruts along which we roll too contentedly all our lives !

We know that the liver is an elastic organ, and that, when there is over-distension of the right auricle and the great veins, it may rapidly enlarge, till it is double its normal size, and then subside again when the heart recovers itself, and the engorgement of the right heart, &c., is lessened.

We know that the liver is elastic, and distends when overfilled with blood, but we never thought of examining to see whether the elasticity of the liver did not lead it to relax and diminish in volume when it was under-filled with blood, owing to the weakness of the heart's action, or other cause.

I am surprised to think that, during the first twenty-four years that I carefully studied the abnormalities due to disease in the human mechanism, I never (until the year 1907) made this simple deduction. I, in common with my brother practitioners, constantly noticed marked diminution in the amount of liver dullness ; often found the intestinal resonance well above the level of the costal arch on the right side, and yet passed the observation by, because I did not know the meaning of it, and did not take the mental trouble of saying : 'Why is the area of liver dullness lessened ?'

I now know that diminution in the area of the liver dullness is a most valuable sign of heart weakness.

The principle underlying this diminution in the size of the organs, we have referred to, may be stated as a general proposition.

Marked variation in the actual volume of the heart and of the more vascular of the elastic solid viscera, both as regards increase or decrease, occurs as a result of variation in the amount of work which the heart is doing, or in the amount of blood which is being kept in circulation by it.

Where the actual size of these organs can be determined by clinical methods, valuable information as to the state of the heart and circulation can thereby be gained.

There is, I think, little doubt that there will be some variation in the size of the spleen, and possibly also of the kidneys; but as their size is not easily definable by our present clinical methods, any variation in their size is of no value to the clinician.

PERCUSSION OF THE LIVER

For the detection of changes due to variation in the size of the liver it is best to map out, by means of percussion, the area of absolute liver dullness, with the patient recumbent on his back. It is also interesting to follow the outline of the relative dullness by means of firm percussion, where it underlies the edge of the lung, and light percussion where it overlies the resonant intestine.

When mapped out in this position, it is not infrequent to find that there is no absolute dullness at all, internal to the right nipple line—the normal dullness of the left lobe of the liver disappearing, as well as most of the anterior part of the right lobe. In such a case, instead of the liver dullness reaching to the costal arch in the nipple line, and crossing across the middle line below the xiphoid cartilage, there will be full intestinal or gastric resonance for some 1½ or 2 inches above the costal arch, and the gastric resonance will meet the lung resonance in the middle line (*vide* figs. 51 and 55).

There are probably four factors which may aid, in varying degree, in producing this disappearance of the liver dullness anteriorly.

(1.) In the first place, there is shrinkage in the absolute volume of the liver, due to abnormal emptiness of its veins and arteries. Judging by the mechanism whereby the liver enlarges when overfull of blood, the hepatic vein, aided by the portal vein, will play a more important part than the arterial system.

(2.) A second factor plays quite as important a part as the one just mentioned, and probably a more important one—namely, the loss of firmness which results from its insufficient distension with blood.

When once the elastic nature of the liver is grasped, this flaccidity of the under-filled organ is easily understood.

When the patient is lying on his back, this flaccidity probably causes the anterior edge of the liver to fall away from the chest wall, and so enable the air-containing intestines to come in contact with the ribs above the costal arch. In this connection it must, however, be remembered that when the edge of the liver is thin, it is not easy by percussion to distinguish it from underlying air-filled intestines.

It is sometimes quite easy to demonstrate this flaccidity. Thus, take a patient of this type, where there is no absolute dullness internal to the right nipple line, when on his back, and then make him sit up and lean well forward. It will then be found that the liver dullness occupies its normal situation anteriorly, but that there is intestinal resonance posteriorly, where liver dullness ought to be (*vide* Case VI, fig. 55).

(3.) The third factor is that to which I referred in the essay on the Rise of the Diaphragm—namely, the altered relationships of the liver anteriorly, which result from a rise in the level of the diaphragm.

With a normal liver and a normal diaphragm, the anterior edge of the liver will reach the costal arch in the right nipple line, and as far down as the seventh rib. When, however, the arch of the diaphragm rises, owing to a shrinkage of the thoracic viscera, the posterior fixation of the liver prevents its rising up into the arch of the diaphragm as a whole, and therefore the loose anterior margin and the left lobe have to rise out of proportion to the rest of the liver, and consequently do not reach as low down anteriorly as formerly, being drawn back, as it were, under the diaphragm. In the case of a very high diaphragm, it is conceivable that the left lobe of the liver, and the anterior margin of the right lobe, may not be able to reach the anterior chest wall at all, being entirely under the arch of the diaphragm.

This third factor does play a very important part, combined with the others (especially the first), in the production of the more extreme cases of disappearance of the liver dullness anteriorly. Nevertheless, a very considerable loss of liver dullness without much evidence of a rise in the diaphragm is by no means infrequent.

(4.) The fourth factor[1] is the altered conductivity to percussion vibrations, which appears to accompany relaxation of the liver tissue. It is extremely probable that the main reason for the disappearance of the liver dullness when the organ is underfilled with blood is that when it is sufficiently flaccid the liver tissue loses its power of damping down the resonance of the underlying stomach and intestines. Over-distension of the liver tissue with blood certainly has this effect, and if it could be proved that under-filling and flaccidity also increased the conductivity of the liver tissue to the percussion vibrations of subjacent resonant viscera, it would explain the regular and sometimes extreme variations in the area of absolute liver dullness far better than either of the foregoing theories. It is sometimes possible to note a complete disappearance of the absolute liver dullness as far back as the anterior axillary line. In such a case the intestinal resonance, on firm percussion, comes right up to the lung resonance as far out as the anterior axillary line. It is inconceivable that there can be a sufficient diminution in the actual size of the liver to account for this phenomenon, and it is not easy to conceive how it could be due to a falling away of the liver tissue from the chest wall owing to flaccidity. Moreover, the relationship of the relative to the absolute dullness along the lower edge of the liver does not altogether suggest the falling back of the edge of the liver and the substitution close to the abdominal wall of resonant intestines instead of dull liver tissue. The way the resonance shades off into the dullness suggests altered conductivity as the cause of the intestinal resonance taking the place of liver dullness.

This same explanation is also suggested by the complete disappearance of the dullness due to the left lobe of the liver. This is very often noticed in heart weakness or malnutrition, and it seems more likely that it results from altered conductivity rather than that the left lobe so diminishes in size as to be able to fall back completely under the arch of the diaphragm.

---

[1] This fourth factor was only realised as a possibility by the writer in December 1913.

Moreover, if the disappearance were entirely due to a falling away of the liver when the patient was lying back, a reversal of his position would not only restore the liver dullness, but also give an abnormal amount.

On making this observation it is found that sometimes the liver dullness returns anteriorly when the liver is percussed while the patient is on his hands and knees, and at the same time disappears posteriorly—as would be the case if flaccidity were the main cause of the disappearance of the liver dullness. In this attitude, the falling forwards of the flaccid liver would restore the dullness anteriorly and allow the resonant intestines to reach the ribs posteriorly.

On the other hand, this experiment does not always give this result and often the increase in the dullness anteriorly is by no means as great as would be the case if the disappearance had been due to flaccidity of the liver tissue alone.

If this theory of altered conductivity proved to be correct, the following generalisation can be made.

The power which the liver tissue possesses of damping down the resonance, which underlying air-containing viscera would give on percussion, and thus giving rise to the dull note which is called the absolute liver dullness, depends upon the tension of its tissue as well as upon the thickness of tissue which overlies the resonant organ.

This power of damping down percussion vibrations and giving a dull percussion note is lessened whenever the tension of the liver tissue is either increased or diminished beyond a certain point. In other words, when the liver tissue is more tense or more flaccid than normal its power of conducting percussion vibrations is increased, and it seems as if the increase of conductivity were proportional to the extent of the departure from the normal tension of the liver tissue as regards both excess and defect.

If this be so, it can be asserted that the liver loses to some extent its power of damping down the resonance of the underlying viscera when its normal tension is either sufficiently increased by over-distension of its vessels with blood or lowered by their under-filling.

The extent of the over-filling and congestion can be estimated by the extent to which the area where the liver gives an absolutely dull note retreats back from the edge of the liver as determinable by palpation.

Also the extent of the under-filling of the liver with blood can be estimated by the extent to which the area of absolute dullness diminishes, and its return towards normal conditions by the return of the absolute liver dullness towards its normal size.

The careful study of a long series of cases—such as I have not yet had time to make—will probably make it possible to differentiate between the part played by each of these three factors in any particular case.

Changes due to flaccidity are, as already pointed out, distinguishable by the effect of change of position. Further, so far as my present observations go, I can make the suggestion that the presence of some dullness, absolute or even relative, in the situation of the left lobe of the liver, together with a narrow strip of dullness joining it with the rest of the liver dullness on the right side, would suggest rather the presence of relaxation of the liver tissue than actual shrinkage —with or without high diaphragm. In cases where the liver dullness is absent in and to the left of the middle line, there is almost certainly shrinkage with or without high diaphragm.

We may learn something as to the condition of the liver, so far as the presence or absence of fibrous tissue is concerned, when we come to know more as to the meaning of the very various shapes which we find that the liver dullness assumes when diminishing in volume owing to circulatory changes.

I need hardly say that I exclude from the scope of these remarks any cases where there is a probability that the lessened size of the liver is of organic origin, and due to cirrhosis or other definite lesion. In the cases upon which this paper is based the shrinkage was of transient nature, lasting only a few days or weeks.

DIMINUTION IN THE AREA OF THE LIVER DULLNESS DUE TO DEFICIENCY IN THE TOTAL VOLUME OF THE BLOOD

When dealing with changes due to deficient distension of the organs with blood, it must be remembered that a circulatory failure of this nature may not only result from weakness of the pump: it will also result from a deficiency in the liquid pumped.

We therefore see precisely the same changes in the liver, as those just described, in cases where the heart is not at fault, but where, as in the case of severe hæmorrhage, there is a lessening in the total volume of the blood, and, consequently, the capacity of the whole vascular system must be lessened in order that the proper blood pressure may be maintained within it. Such a lessening of the total volume may also take place, as clinical observation proves, owing to a deficiency in the body of some of the essential constituents of the blood, for in the presence of such a deficiency the total volume must be compensatorily diminished, in order that the proper percentage composition may be maintained. The simplest illustration of this is the diminution in the volume of the blood which takes place in starvation.

The causes of the shrinkage of the liver and other organs may be thus summarised :—

Deficient fullness of the vascular system, resulting in shrinkage of the more vascular of the elastic solid viscera, results from—

I. Failure in the muscular power of the heart.

II. Deficiency in the total volume of the blood, due to—

1. Actual loss of blood from hæmorrhage.
2. Loss of water from the body—as in cholera, &c.
3. Failure of the blood-forming organs, or excessive blood destruction, &c., causing anæmia.
4. Compensatory diminution of the total volume of the blood in order to maintain at its proper percentage some essential constituent of which there is an insufficient amount in the body, for instance—

(*a*) Deficiency of ordinary proteids and nutritive materials, as in any form of starvation.

(*b*) Deficiency of some internal secretion, such as that of the thyroid gland.

(*c*) Deficiency of special materials necessary for the nutrition of the nervous system.

(*d*) Defective volume of the blood seen in cases of neurasthenia without malnutrition.

In addition to these causes of variation in the extent of the liver dullness, it must not be forgotten that in a very small percentage of apparently healthy persons the diaphragm may be above the normal line, or the area of the liver dullness may be abnormally small. I have not as yet, however, met with more than four or five such instances.

The clinical aspects of diminution in the liver dullness, due to myocardial weakness, will first be dealt with.

I

DIMINUTION OF LIVER DULLNESS DUE TO MYOCARDIAL WEAKNESS

If the area of liver dullness and the outline—especially the lower border—of the cardiac dullness be watched carefully in cases such as acute rheumatism, where there is risk of weakening of the myocardium by toxæmia or inflammation, some very instructive observations may be made, and the value of this physical sign to which I am drawing attention will become apparent.

The following is such a case where a careful record of the area of the liver dullness and of the cardiac dullness showed most interesting changes taking place from day to day.

I give a large number of figures showing the changes that took place in this case; for they are all more or less characteristic of what may be found in cases of cardiac weakness with and without dilatation of the right ventricle, and also in cases where the liver dullness is diminished from circulatory causes other than cardiac weakness (see figs. 42–52).

CASE I.—The patient was a young man aged about twenty. When first seen, he had been under treatment for ten days for

Case Showing Variations in the Size of the Liver Dullness due to Heart Weakness

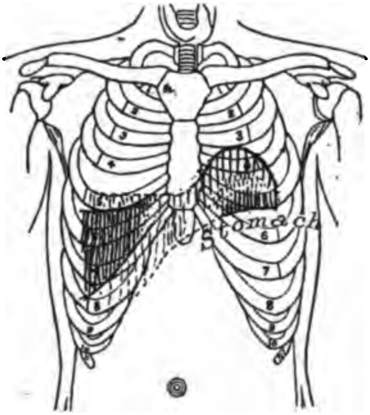


Fig. 42.—Case I.

May 16. Acute rheumatism. Temperature 102° F.; pulse 110. Liver small; diaphragm high.

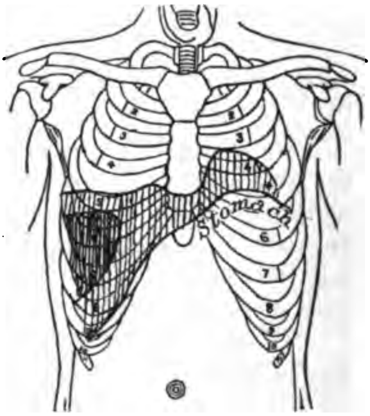


Fig. 43.—Case I.

May 18. Much better after full doses of sodium salicylate, but diaphragm higher and heart and liver smaller.

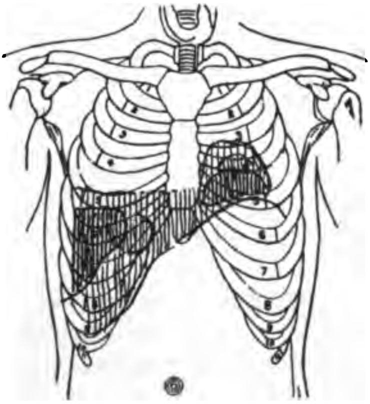


Fig. 44.—Case I.

May 20. Still improving. Heart larger; diaphragm lower.

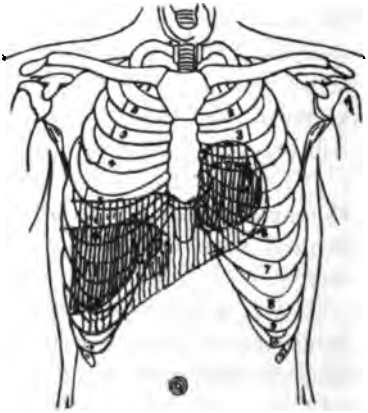


Fig. 45.—Case I.

May 23. Better. Heart, liver, and diaphragm nearly normal.

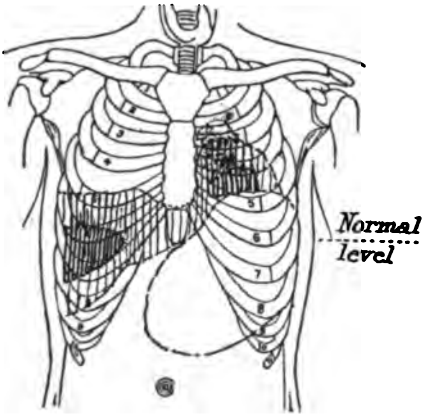


FIG. 46.—CASE I.

May 26. Heart stronger, but the thin part of the right ventricle is giving way. Diaphragm rising again.

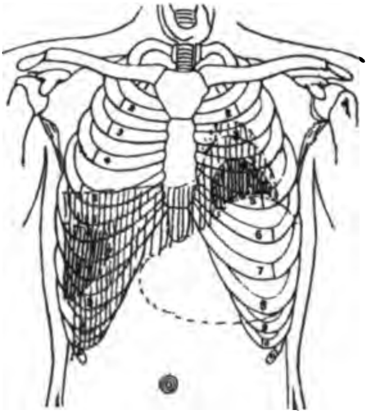


FIG. 47.—CASE I.

May 29. Conus of right ventricle dilated, but liver larger and diaphragm lower.

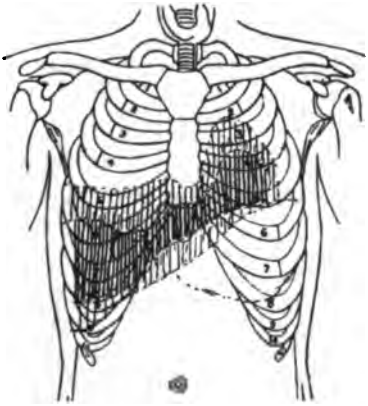


FIG. 48.—CASE I.

June 1. Right ventricle more dilated, but liver larger and diaphragm lower. Patient better.

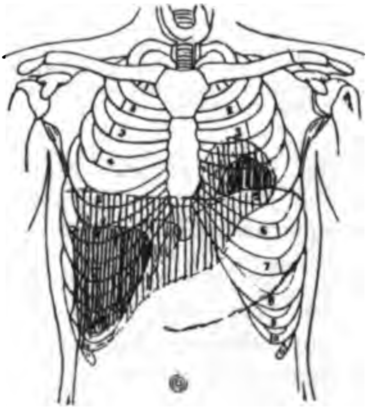


FIG. 49.—CASE I.

June 7. Heart smaller, but patient not so well: has rheumatic pain again.

acute rheumatism, by ten-grain doses of sodium salicylate. To this he had not properly responded, for the joint pain and swelling was still considerable, and the temperature at times reached 102° F. The pulse was 100–110. There was mitral regurgitation of considerable extent, for the murmur was conducted down the spine to the sacrum; the apex was external to the nipple line, but otherwise the heart was not dilated or enlarged.

The diaphragm was high, and the absolute liver dullness did not reach the costal arch. This was on May 16, and the size of the cardiac and hepatic dullness are shown approximately in fig. 42.

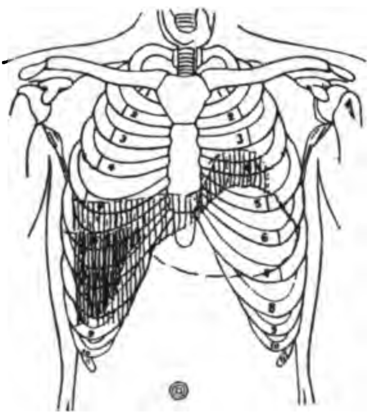


FIG. 50.—CASE I.

July 22. Heart and liver dullness smaller and diaphragm higher again. Fresh endocarditis.

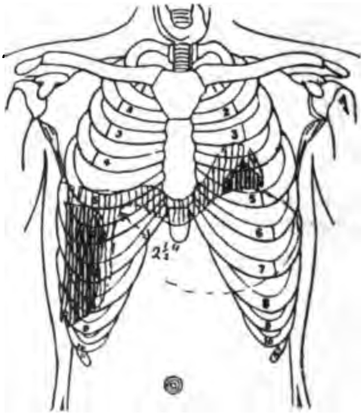


FIG. 51.—CASE I.

July 27. Heart larger, liver dullness smaller. Full doses of sodium sulphocarbolate and quinine.

The subsequent diagrams were made—usually by the bed-side—after carefully percussing out the areas of absolute and relative dullness and outlining them on the chest. For such work as this, the 'zinc dust and aluminium or silver spatula' method is preferable to aniline or other pencil, from both the patient's and the physician's point of view.

If the skin be rubbed with zinc and starch dust, it is then possible to mark it with any silver, aluminium, or german-silver spatula or letter-opener—just as if one were writing on paper with a lead pencil. I am indebted to the father of this particular patient for working out the explanation of this phenomenon, and I do not think it has hitherto been published. I was not aware of the explanation when I described this method of writing on the skin some years ago. It is apparently purely a mechanical phenomenon; the particles of zinc oxide detach particles of the metal, giving what is technically known as 'a streak,' just as one can write on stone with a piece of metal. The skin can also, it appears, be rendered capable of receiving a black mark from a soft metal point if it be rubbed with such a material as asbestos.

I have heard of this property of the skin—of taking impressions from the softer metals—being mistakenly associated with trophic changes occurring in paralysis of various kinds, because the clinical teacher in question did not discover that the dermographic power was not a function of the skin, but of the 'ward-dust' which was applied to the skin of the paralysed parts by the nurses in charge of the case.

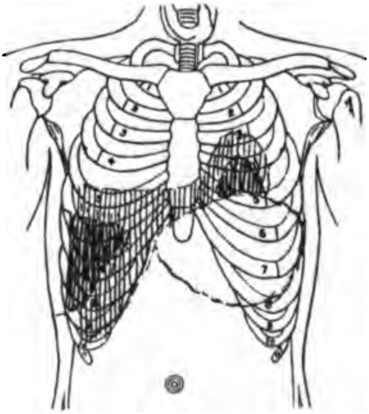


FIG. 52.—CASE I.
August 15. Patient much better again.

But to return to the patient. The diagrams, as will be seen, were taken at first every two or three days, and subsequently at longer intervals.

Fig. 43 shows a decided lessening of the size of the cardiac and liver dullness, with a rise in the diaphragm of an inch or more, as judged by the level of the gastric resonance. This may have been due to the continued action of the rheumatic poison, or it may have been due to the weakening of the heart caused by full doses of sodium salicylate: for full doses are often badly borne when small doses have been given for some time previously. In this case the ultimate result was good, and the next two figures show the increasing size of the heart and lungs. Then in fig. 46 there is a marked change, which is at first difficult to explain. The heart had been steadily improving in the strength and precision of its sounds, and they were now sharp and good; the patient also was feeling better. Why, then, the rise in the diaphragm and the great diminution in the liver dullness which this diagram shows?

Another change is also shown—namely, upward increase of the cardiac dullness, which was combined with the appearance of pulsation in the second and third left interspaces. This gives, I think, the explanation.

With the elimination of the rheumatic toxin, the muscular power of the heart had increased, but the tissues of the heart were still much below their normal strength. As a result, the weakest part of the right ventricle—namely, the so-called 'conus arteriosus'—being unable to stand the rise in the blood pressure, had begun to dilate.

Nature had in consequence—by lessening the volume of the blood in circulation—endeavoured to lessen the strain on this weak portion. Hence the rise of the diaphragm and the lessened hepatic dullness.

The next two figures (47 and 48) show continued improvement, and in the latter, which was taken on June 1, the relationships are very near the normal. A week later, there was a little return of rheumatism, calling for an increased dose of salicin and accompanied by a diminution in the area of liver dullness (fig. 49).

By the end of June, the patient was much better and beginning to walk about a little.

This improvement was, however, soon followed by a relapse. The heart became more rapid again; the temperature rose slightly. An aortic diastolic murmur, which previously had been faint and indistinct, was now well marked, showing a recurrence of the endocarditis.

This relapse commenced on July 13, and was well marked on the 22nd, when fig. 50 was taken. Figs. 50 and 51 both show again a marked diminution in the size of the cardiac dullness with a rise of diaphragm and diminution of the liver dullness.

On July 22, the date when fig. 50 was made, he had been on sodium sulpho-carbolate (gr. xx) every two hours, together with quinine (gr. ij) every two hours alternately. At this time, he was beginning to show some physiological symptoms of the action of each of the drugs, and the frequency of the dose was therefore cut down to every three hours, with some omissions during the night. On this dose he remained till his recovery.

Fig. 51, taken after being on the sulpho-carbolate and quinine for ten days, shows a remarkable diminution in the left lobe of the liver without any great change in the right.

Fig. 52 shows the condition of heart and liver on August 15, at which date he was rapidly gaining weight and all fear of his endocarditis developing into septic endocarditis was at an end, and he was on the way to a most satisfactory recovery and with a heart but very slightly damaged.

The following is another instance of the important information given by diminution in the size of the liver, but from the opposite extreme of cardiac failure as compared with the last case.

Case II.—The patient was a lady of forty-eight years, with mitral regurgitation and a weak and dilated heart following influenza, with general anasarca threatening, and a liver down 2 inches below the costal arch from venous engorgement.

One day it was found that the liver was 1½ inches smaller than it had been two days previously. Was this a sign of improved circulation? It might have been; but here it was not so, for the patient was rather worse if anything, though not markedly so. It was a sign of failing heart muscle and a call for stimulation—albeit, very careful stimulation, owing to the faulty nutrition of the heart muscle. In a couple of days the liver had returned to its former size, and the threatened danger of heart failure had been averted. Had the variation in the size of the liver not given timely warning, valuable time might have been lost before the treatment was changed, for the importance of the slight alteration in the patient's symptoms might quite easily have been overlooked (see also p. 63).

Instances could easily be multiplied of the value of diminution in the size of the liver dullness as indicating cardiac failure, and it is specially so in cases of myocardial weakness and degeneration. The following is an instance:—

Case III.—The patient, a man aged fifty-six, had suffered for many months from heart weakness and persistent high tension, the sphygmomanometer showing a blood pressure of 210 mm. of mercury, as a rule. One day, he over-exerted himself physically and mentally, and, shortly after reaching home, became unconscious and had a slight amount of right hemiplegia. The pulse was weak and rapid, and the breathing irregular and almost 'Cheyne Stokes' in character.

Was the case one of cardiac failure or of cerebral thrombosis?

On examining the heart there was almost no cardiac dullness of any kind to be detected, and an extremely feeble impulse. There was no fullness of the neck veins and no absolute liver dullness in the middle line, and very little internal to the right nipple line. Also the intestinal resonance could be obtained for some 2 inches above the costal arch where the liver dullness ought to have been.

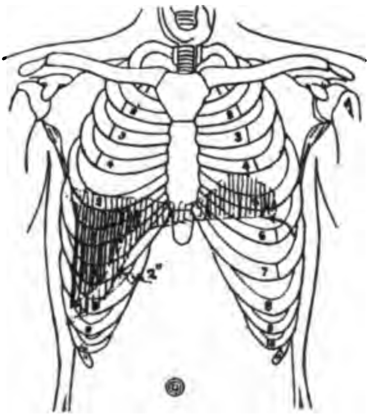


Fig. 53.—Case III.

Small heart and liver dullness. Heart failure in arterio-sclerosis.

The accompanying diagram (fig. 53) represents with fair accuracy what was found, although the patient was too ill to admit of accurate notes being taken at the time. It will be noticed that the gastric resonance reached to the fifth rib, showing a rise of the diaphragm on the left side. On the right it was within the normal limits.

This examination of the liver showed clearly that the cardiac condition was primary, and the circulatory failure in the brain secondary. He was treated as a case of myocardial failure, and the brain condition rapidly improved. In a week or so the heart gained strength and the liver dullness increased in volume reaching to the costal arch and being also absolute in the middle line. The cardiac dullness also increased in size.

II

CLINICAL ASPECTS OF DIMINUTION OF THE LIVER DULLNESS DUE TO A SHRINKAGE IN THE TOTAL VOLUME OF THE BLOOD IN ACTIVE CIRCULATION

The clinical aspect of cases where the scantiness of the circulating blood does not depend upon the heart, but upon an actual deficiency in the blood itself, must now be briefly discussed.

1. Small liver in hæmorrhage.—Firstly, where that deficiency is due to actual hæmorrhage, the emptiness of the vascular system is easily understood, although I am not so sure that the effect of this emptiness upon the heart, lungs, and liver is generally recognised.

The following is a good illustrative case:—

Case IV.—Nurse A., suffering from enteric fever, had a sudden severe hæmorrhage amounting to some three pints of blood or more. After the hæmorrhage, it was found that the liver dullness was greatly diminished and that intestinal resonance was obtainable for some 1½ inches above the right costal arch. After the subcutaneous administration of saline solution, the liver dullness increased again in area, and at the end of thirty-six hours was back again at the costal arch.

No more need be said on this subject, and the examination of almost any case of severe hæmorrhage will show the diminution in the liver dullness here described.

2. It will be easily understood that a similar state of things will result from the dehydration of the body which results from profuse diarrhœa and vomiting.

3. In some cases of anæmia, the condition of liver and diaphragm point to the fact that the total volume of the blood is lessened and that the vascular system is badly filled; but the relationship of high diaphragm and smallness of the liver to anæmia is somewhat complicated, and I have not yet had the opportunity of working out the solution of the problem thus presented.

4. In compensatory diminution of the volume of the blood.—In yet another group of cases where smallness of the liver dullness is well marked and is sometimes accompanied by high diaphragm as well, it seems that the total volume of the blood in active circulation has been cut down by some compensatory mechanism because of a deficiency within the body of some of the essential constituents of the blood.

Small Liver in Starvation, &c.

(*a*) The simplest instance of such a deficiency occurs in the case of a deficiency in the absorption of ordinary nutritive materials, proteids, &c., such as occurs in simple starvation.

The effect of starvation can easily be observed, clinically, where cases of gastric ulcer are fed by the bowel only. Careful percussion of the stomach and liver in such a case will, as a rule, show that day by day the level of the gastric resonance rises (showing a rise of the diaphragm), and the area of liver dullness diminishes owing to the increasing emptiness of its blood-vessels. A series of such observations is given in Essay II, at p. 170, where six figures are given—namely, figs. 25–30, showing the changes which took place on diminishing and on increasing the amount of food given.

Small Liver Dullness due to Malnutrition

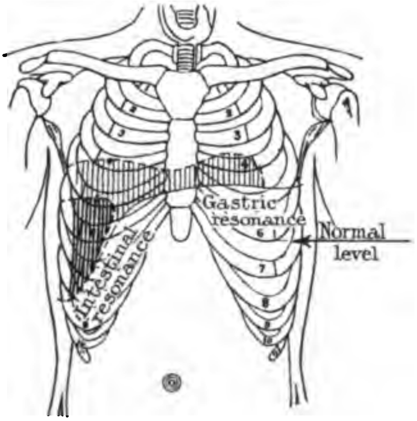


Fig. 54.—Case V.

A case of gastric ulcer, showing small liver dullness after six days' rectal feeding.

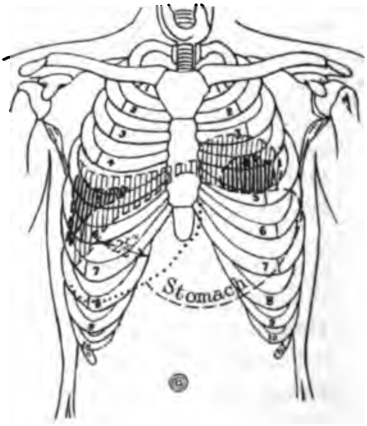


Fig. 55.—Case VI.

Very small liver dullness and very high diaphragm in malnutrition from pyloric narrowing.

The dotted line shows the edge of the absolute liver dullness when the patient sits up and leans forward.

I here reproduce fig. 26, showing the extreme reached after six days of rectal feeding (*vide* fig. 54).

A similar change may also be sometimes seen in enteric fever, where a patient is sometimes brought very low owing to the interference with intestinal absorption which results from the inflammatory swelling of the mesenteric lymphatic glands.

In this group of cases the careful observation of the size of the liver and the level of the diaphragm gives most valuable information as to the degree in which the general nutrition of the patient is effected.

A well-marked case of diminution in the area of the liver dullness, due to defective nutrition, is the following (*vide* Case VI, fig. 55).

W. O., aged forty-six, had suffered for very many years from indigestion, consisting of severe pain coming on a couple of hours after meals. Latterly, the pain had been so troublesome that he had taken very little food, and he had, moreover, periodic attacks of vomiting. Examination of the abdomen showed that he was suffering from pyloric stenosis with compensatory hypertrophy of the gastric muscles and no material amount of dilatation. The man had been living an outdoor life in South Australia, and working hard.

The liver dullness was extremely small as he lay on his back, and its absolute dullness began at the upper border of the fifth rib in the right nipple line, but there was intestinal resonance in the fifth interspace. The absolute dullness did not reach inwards much beyond the nipple line, and in the axilla its lower limit was very much higher than normal.

In the middle line the full gastric resonance reached up to the level of the fourth interspace, and there were only some 1½ inches of relative dullness separating the gastric from the lung resonance.

The part played by flaccidity of the liver in this case was well shown by the fact that when the man sat up and leant well forward, the liver dullness returned to its normal situation anteriorly, and there was nearly or quite the usual area of dullness in and internal to the nipple line; but the liver dullness disappeared from the back, and the intestinal note could be got up as far as the lower level of the lung resonance about the tenth rib. There was no absolute dullness at the back, behind the mid-axillary line when the man was in this position. There was a little relative dullness; but over the greater part of the area usually occupied by the liver dullness there was full intestinal resonance.

SMALL LIVER IN MYXŒDEMA

(*b*) The next group of cases is less simple in their causation than the last, and less easily understood, but they form an intermediate group between the last group and the next, which is more complex still.

In this group it seems as if the deficiency of some important internal secretion led to a compensatory limitation of the volume of the blood in circulation.

SMALL LIVER DULLNESS IN NEURASTHENIA

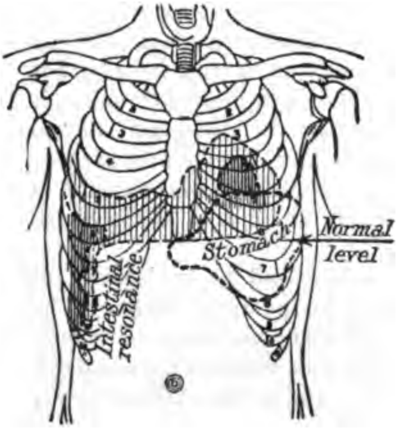


FIG. 56.—CASE VII.
Small liver dullness in myxœdema.

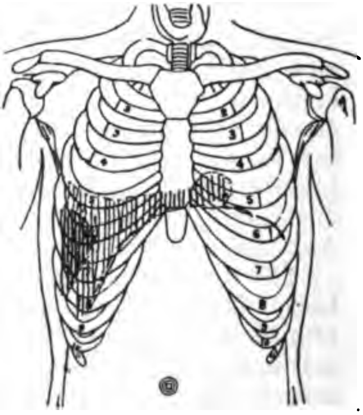


FIG. 57.—CASE VIII.
Small heart and liver dullness with high diaphragm in neurasthenia.

The following is such a case which was described in the last essay, although the theory there given is inadequate to explain the clinical picture.

CASE VII.—The patient was a labourer who either could not or would not work, and constantly complained of lassitude. The man was to all appearances in perfect health, suffering from nothing more than a little dyspepsia. Careful physical examination, however, showed a very high diaphragm (on the left side mainly) and also a most marked diminution in the area of the liver dullness, as is shown by the diagram (fig. 38), which I here reproduce (*vide* fig. 56).

This smallness of the liver dullness and high gastric resonance suggested some fault in his circulation which would account for his weakness. None was, however, found. In spite of his colour being good, there was something in his appearance and his symptoms which suggested myxœdema. Thyroid extract was therefore tried, with the result that it entirely cured him. I am sorry, however, that I did not take a second diagram to demonstrate the contrast.

SMALL LIVER IN NEURASTHENIA

(c) In the last group with which I have to deal, the fact of high diaphragm and small liver dullness can be noted in association with certain symptoms of weakness and incapacity suggesting compensatory diminution in the volume of the blood. Often in such cases, by means of treatment along general lines, there are evidences of improvement in the circulation, with the return of the liver and diaphragm to their normal relationships; but no light is shed upon the nature of the substance which was presumably deficient in the blood, and which caused a compensatory diminution in its volume.

It is especially in certain cases of neurasthenia that this smallness of the liver can be found. In such a case it is presumably some one of the substances necessary for the proper nutrition of the nervous system which is deficient, and which under treatment and rest gradually re-accumulates in the system until its normal percentage is once more restored.

CASE VIII.—The following is such a case. Mrs. B., aged twenty-six, came to me Feburary 1906, some months after the birth of her first child, complaining of distressing fancies of various kinds, which worried her much, and which she could not shake off. They made her so self-conscious that she could not go out into society. She also had most troublesome salivation. She was to all appearances in perfect health, and very well nourished, with no trace of anæmia or dyspepsia or other ailment. On examining the liver dullness, it was found to be remarkably small. There was no absolute dullness internal to the right nipple line, and the relative dullness did not come near the costal arch. There was no absolute cardiac dullness, and the relative dullness would be covered by a half-crown piece. The stomach resonance was up to the fifth interspace (*vide* fig. 57). In spite of her healthy appearance and good colour, it was therefore probable that the total volume of her blood was small. On inquiry, it transpired that she had eaten very little meat throughout her pregnancy, adopting largely a vegetarian and fruit diet. It seemed probable that this had not been sufficient for her, although its proteid content seemed fairly good. She was put on full meat diet with a little thyroid extract, and in four weeks her liver dullness had returned nearly to the normal size—the absolute dullness reaching to the costal arch—and she had much more control over her thoughts. She continued steadily to improve. Here the size of the liver dullness gave the clue to the cause of the neurasthenia and its treatment.

This smallness of the liver, with or without a high diaphragm, will very frequently be found to occur associated with neurasthenia.

It is, in my opinion, probable that all cases of neurasthenia may be divided into two classes—distinct both as regards cause and treatment—by the presence or absence of this symptom.

Enough has now, I think, been said to show the importance of the little-known physical signs which form the subject of this essay.

Essay IV.—ON SOME DIAGNOSTIC DIFFICULTIES ASSOCIATED WITH DILATATION OF THE RIGHT VENTRICLE[1]

For the purposes of this essay it will be convenient to divide cases of dilatation of the right ventricle, clinically, into three main groups.

Firstly.—Cases where the tricuspid valve becomes incompetent, and the regurgitation which occurs takes place mainly upwards into the superior vena cava and its branches, thus giving rise to pulsation in the veins of the neck.

Secondly.—Cases where the tricuspid incompetence mainly takes effect downwards into the liver, and there is no evidence of regurgitation of blood upwards into the veins of the neck.

Thirdly.—Cases where tricuspid regurgitation does not necessarily play an important part in the clinical features of the case, but where the dilatation mainly involves the upper part of the anterior wall of the right ventricle—i.e., the so-called conus arteriosus.

The first two of these three types most commonly occur secondarily to valvular disease, whereas the best marked cases of the third type are seen in the dyspnœa of anæmia and cardiac overstrain.

Although each of these types of right ventricular failure often occurs more or less independently of the others, no hard-and-fast line is to be drawn, for they may each or all occur simultaneously or successively in any particular case of cardiac failure. As a general rule, the third type of right ventricular failure is relatively more common in youth and adolescence than it is later in life.

I shall only refer briefly to the second of these three groups, and shall therefore take it up first. I shall then consider the third group, and shall take up the first group last.

---

[1] See *Birmingham Medical Review*, June 1900.

In the second group the tricuspid reflux mainly expends its force in dilating the lower and less muscular portion of the right auricle and in distending the liver.

In these cases it seems as if the regurgitating blood were prevented from passing upwards into the veins of the neck by the satisfactory muscular tone of the right auricle and of the veins, and that, as a consequence, the lower part of the auricle and the liver had to bear the brunt of the regurgitation. It is possible, however, that the reverse of this is the true sequence—namely, that the veins of the neck escape because the Eustachian valve gives way early in the case.

Clinically, it is by no means uncommon to see cases where the liver is so enlarged that its lower edge is at or below the level of the umbilicus, and where the organ can be felt to pulsate from distension at each beat of the heart, but yet no pulsation is observable in the veins of the neck. Two such cases I have seen within the last week or two, and I have the notes of an extreme case where there was an aneurysmal dilatation of the lower part of the auricle and of the part of the inferior vena cava above the diaphragm. In this case the cavity of the right auricle held 570 c.c., or exactly 20 ounces, when moderately distended; and of this about 8 ounces, as far as could be estimated, was the content of the aneurismal dilatation above referred to. The physical signs in these cases do not leave much room for doubt as to the diagnosis, and I shall therefore pass on to the other two groups.

I shall now speak of difficulties in diagnosis included in the third of my three groups—namely, where the right ventricle dilates upwards in the manner so fully described by the late Dr. Foxwell in his Paper on 'The Heart in Anæmia and Debility,' in the *Lancet* for the year 1891, in 'Essays in Heart and Lung Disease,' and most recently in the Bradshaw Lecture, 1899.

In this type of dilatation, the following difficulties may arise:—

1. The right ventricular pulsation, which is visible and palpable in the second left interspace, may be mistaken for that of an aneurysm of the transverse arch of the aorta, or vice versa. Cases are, I believe, on record when the former error was made, and I shall recount later on a case where it was by no

means easy to distinguish an aneurysm of the aorta from a dilatation of the right ventricle.

2. Another difficulty, to which I shall refer, is that which sometimes occurs in distinguishing the systolic pulmonary murmur, which results from this type of dilatation, from a murmur of aortic origin.

Before taking up these two points it will perhaps be well to say a few words as to this upward dilatation of the ventricle. In this type of dilatation, as I have previously stated, tricuspid regurgitation does not play an essential part, and the tricuspid valves may be, and I believe often are, quite competent. This upward dilatation seems to result when the work to be done by the contracting ventricle exceeds the resisting power of the weakest part of its wall. When this state of matters occurs, the ventricle wall will bulge at the weak spot. This may be well illustrated by grasping a partly filled rubber toy balloon. When this is done, the portion outside the hand will be made to bulge by the compression of the part within the grasp of the hand.

In the case of the right ventricle, the lower part of the ventricle is the strongest. When the resistance to be overcome in the pulmonary artery exceeds the resisting power of the thin portion of the anterior wall which lies just below the pulmonary valves, this weaker part will be made to bulge upwards and forwards with each contraction of the ventricle. This upward bulging is often so considerable that the pulmonary valves come to lie under the second rib or even higher, and in such cases the systolic pulsation of the right ventricle may be seen and felt in the second left interspace. This pulsation may be mistaken for that of an aneurysm, or vice versa.

Without going at all fully into all physical signs of this dilatation, I must nevertheless dwell for a short time on those which bear upon my present subject. They are:—

(*a*) Increase of the heart upwards, giving rise to a considerable amount of relative cardiac dullness in the second left interspace and sometimes to absolute dullness also. In addition, there is considerable increase of the cardiac dullness in the third left interspace.

(*b*) Systolic pulsation in the second left interspace.

(*c*) A well-marked systolic murmur heard over the area of dullness above mentioned; and in extreme cases a well-marked systolic thrill. The point of maximum intensity of the murmur is not infrequently an inch or more away from the sternum, just as might be expected in a case of aneurysm.

(*d*) There is also, sometimes, a considerable amount of tenderness and of pain on exertion over the pulsating area.

(*e*) Some patients also complain of a choking feeling in the throat—as if some food had stuck in the throat opposite the upper end of the sternum. This, too, is a symptom of aneurysm of the transverse arch of the aorta.

To give an instance: I have seen a young man about thirty, who, when convalescent from an attack of pneumonia, ran nearly half a mile to escape a threatened shower of rain. On reaching his destination, he was much out of breath, and was alarmed somewhat at remaining so; he also had rather a sharp pain above the left nipple. When I examined him shortly afterwards, I found there was systolic cardiac pulsation in the second left interspace with a systolic murmur. There was considerable tenderness in the second left interspace on percussion, and the patient complained of a dull aching pain in the same situation, which was worse on exertion, and also of a sensation in the throat—such as I have just referred to.

In this case, I had no difficulty in diagnosing acute dilatation of the right ventricle from overstrain; but had the case been rather more severe it would not have been so easy to come to a diagnosis. I have often seen similar symptoms in apparently healthy young men as a result of rowing or other form of excessive muscular exertion.

Although I have not yet seen a case of dilatation of the right ventricle where I could not on careful examination certainly exclude aneurysm of the aorta, I have seen a case of aneurysm of the aorta in a young woman where, on my first examination, I found it impossible to diagnose the condition from dilatation of the right ventricle. A second examination, however, after the patient had been taking somewhat sharp exercise, sufficed to clear up the diagnosis. But even after I was convinced of the presence of an aneurysm, the symptoms and physical signs were at times so exactly like those of dilatation of the right ventricle that I was once or twice almost tempted to doubt my own diagnosis.

Both before and after the period when the patient was under my care, I believe that she consulted several other physicians, and if I am correctly informed there was a striking lack of unanimity in the diagnosis at which they arrived. Fuller particulars of this case will doubtless therefore be interesting.

The first case I refer to is that of Mrs. P., whom I first saw in October 1897. She was then aged thirty. She was complaining of an intermittent pain in the left pectoral region, which was increased, or brought on, by exertion and sometimes radiated down the left arm. She had had the pain more or less for two or three years. Earlier in life, her occupation had involved a certain amount of hard work and occasional muscular strain. Since her marriage, in 1893, she had lived an easy life. She began to suffer from ill-health soon after her marriage, but she dates her present symptoms from a severe attack of pleurisy in 1895, two and a half years prior to the time when I first saw her. For the last eighteen months she had been much the same as when I saw her. She had a miscarriage soon after recovery from the pleurisy, but there was no history of syphilis discoverable. She was well nourished and of a healthy appearance, and not specially anæmic. The chief complaint was of the pain in her left pectoral region. It was not constant. It would be troublesome for two or three days at a time and then disappear for two or three weeks. It was sometimes bad at night, and came and went like neuralgia. It was, however, increased by exertion, and was accompanied by slight dyspnœa and some palpitation. As to the situation of the pain it would usually commence about the left breast and shoot through to the back and also down the left arm and sometimes to the right arm also. She had been unable to do any work for some months on account of the pain being brought on easily by movements of the arms. She also complained of a sensation of a lump in the throat when she was excited.

As to the Physical Examination of the Chest.—On inspection there was slight diffuse pulsation visible in the second, third, and fourth left interspaces. The apex was under the fifth rib, a little external to the nipple line. The cardiac dullness extended to the second rib, being nearly absolute below the level of the fourth rib. There was no pulsation in the fifth interspace. To the right the heart dullness did not extend beyond the left edge of the sternum. There was no murmur audible at the apex or in the axilla. There was, however, a loud systolic murmur at the base of the heart, heard best over the second left interspace, 2½ inches from sternum. This systolic murmur was audible over an area about the size of the palm of the hand, and in spite of its extreme loudness it was not at all well conducted into the vessels of the neck. The murmur was, however, audible posteriorly. It was loudest about the middle third of the interscapular area on both sides of the spine. It was less loud at the angle of the left scapula, and was not audible as far forward as the left axilla.

I felt unable to arrive at a definite diagnosis at this visit. The physical signs and symptoms would do for either aneurysm of the transverse arch of the aorta or for upward dilatation of the right ventricle from overstrain. The severity of the pain, the duration of the symptoms, and the conduction of the murmur were certainly suggestive of aneurysm. The pulsation was, however, of the ill-defined type so characteristic of right ventricular pulsation as seen in the second left interspace. I omitted on this occasion to make the patient take a little active exertion, or I should have lessened my uncertainty. On a subsequent visit, after a little brisk walking, the pulsation in the second left interspace became much more evident; it was now strong and arterial in type, and from this and other symptoms I had no hesitation in diagnosing aneurism of the transverse part of the arch of the aorta.

By my advice she took to her bed, and had more than a year of absolute rest. After this, having heard something about the X-rays, she became anxious to be examined by their aid, and this was done by Mr. Hall-Edwards in my presence. There was clearly a dark shadow in the region of the suspected aneurysm, which was distinct from the heart, and which pulsated. This left no room for doubt as to the diagnosis. As the patient was now fairly free from pain on exertion and seemed much better, she began to get about rather more, but was still very careful. She seemed to be getting on fairly well till, one day, as the result of some slight exertion, the aneurysm ruptured into a bronchus, and she died suddenly of hæmoptysis.

The points of diagnostic importance in these cases seem to me to be as follows :—

(*a*) The character of the impulse in the second space.

(*b*) Non-conduction of the murmur to the neck to the extent which would be the case if the murmur were a pulmonary artery murmur.

(*c*) Its loudness posteriorly in the interscapular area.

(*d*) The severity of the attacks of pain.

(*e*) A separation of the aneurysmal dullness from the cardiac dullness. This was not present in this case.

(*f*) Examination by the Röntgen rays.

(*g*) In such a case as this the cardiograph is of great value in clearing up the diagnosis.

I need hardly point out that the great difference between both the prognosis and treatment of aneurysm and dilatation of the right ventricle makes an error in diagnosis a serious one in such a case.

I now come to my second subheading—namely, the difficulty which may sometimes arise in distinguishing the systolic murmur which accompanies this type of dilatation of the right ventricle from a systolic murmur of aortic origin. In the slighter cases of upward dilatation of the right ventricle, such as are ordinarily seen in the dyspnœa of anæmia, this difficulty does not arise, because the area over which the murmur is audible is limited to the left side of the sternum with its point of maximum intensity in the second left interspace. In the more severe cases of dilatation which occur where the anæmia is very considerable or where the overstrain has been great, the pulmonary artery murmur may be propagated into the aorta and its branches. The pulmonary artery murmur may thus resemble an aortic systolic murmur. I have notes of many cases where I have heard the pulmonary artery murmur conducted into the subclavian arteries, carotids, and even into the axillary arteries.

A glance at the relationship of the pulmonary artery to the aorta will, I think, show how this conduction is brought about. The pulmonary artery murmur is produced in a dilated pulmonary artery. The dilatation is brought about as follows. The upward dilatation of the right ventricle naturally carries the pulmonary valves up with it. As the normal course of the pulmonary artery is *upwards* its course must be shortened by this upward movement of its origin. This shortening must cause relaxation of its elastic walls, and this relaxation must admit of an aneurysmal bulging of the walls under the systolic blood pressure.

Considering how close the aorta lies above the pulmonary artery, normally, any upward movement of dilatation of the latter must bring it into very close contact with the aorta. This contact is so close that the murmur vibrations of the blood in the pulmonary artery will be able to spread through to the blood in the aorta by contiguity of tissue. Hence the murmur may become audible in the arteries, and does on superficial examination simulate an aortic murmur.

When the possibility of an occasional arterial conduction of the pulmonary systolic murmur is once recognised its diagnosis from an aortic systolic murmur is as a rule comparatively easy. The following are the chief points to be noted :—

(*a*) The point of maximum intensity of the murmur is in the second *left* interspace, often half an inch or more away from the sternum.

(*b*) The murmur is less loud in the arteries of the neck than it is in the second left interspace. This is perhaps the most important diagnostic sign. If the murmur be produced at the aortic orifice it must be heard at least as loudly over the carotid arteries as over the sternum ; for if the blood in the aortic orifice were vibrating sufficiently strongly to produce a murmur which is audible through the sternum and lungs, it is hardly conceivable that the vibrations should have sensibly diminished in loudness during the very small fraction of a second which it takes for the blood to reach the carotid arteries, where only the skin and a little muscular tissue intervene between the vibrating blood and the stethoscope.

(*c*) Another fact which will sometimes afford assistance is that the pulmonary artery murmur is, I think, usually better conducted to the left carotid and subclavian than to the right (possibly, because the point where the pulmonary artery comes most closely in contact with the aorta is higher than the point where the innominate artery is given off). Not infrequently, however, there are other systolic murmurs present in the neck which will prevent this observation from being made. This is especially the case with the systolic murmur, of which I shall speak later, which originates in the superior vena cava, and which is apt to occur in those cases of anæmic dilatation of the heart. It is heard well to the right of the sternum, and in the vessels on the right side of the neck better than on the left side. When it occurs with the pulmonary systolic murmur it will render the diagnosis from aortic disease a little less easy.

(*d*) Other signs of dilatation of the right ventricle will be present, such as increase of the cardiac dullness upwards. Moreover, in cases where the pulmonary systolic murmur is loud enough to be conducted well into the arteries there will in all probability be the typical right ventricular pulsation in the second left interspace, also a degree of anæmia will be present that will be suggestive of the 'hæmic' origin of the murmur.

DIFFICULTIES OF DIAGNOSIS IN CONNECTION WITH THE MURMURS DUE TO TRICUSPID REGURGITATION

The murmurs due to tricuspid regurgitation may give rise to difficulties in various ways; but in order to get a clear idea of them it is well to remember that the stream of blood on regurgitation through the tricuspid valve may give rise to a systolic murmur at three different points during its course from the right ventricle to the veins of the neck. Firstly, at the tricuspid valve itself; secondly, at the point where a distended superior vena cava will be constricted on passing through the pericardium, owing to the non-dilatation of the ring which receives support from that structure; and thirdly, a murmur may much more rarely arise at the valves in the veins themselves, when these first give way in the face of the tricuspid regurgitation.

THE TRUE TRICUSPID MURMUR.—The main difficulty in the diagnosis of this murmur is to decide whether a murmur heard to the left of the sternum is really of tricuspid origin. In cases where the left ventricle is but little enlarged and the greater part of the front of the heart is occupied by the right ventricle, a tricuspid may resemble a mitral murmur; but if the direction of conduction be noted, no error need be made, for if there *is* a tricuspid murmur it will be conducted down and to the right as clearly as is the right ventricle first sound; whereas if the murmur be of mitral origin this will not be the case. The hearing of a first sound over the liver more free from murmur than is the case with the first sound heard just to the left of the sternum is strong evidence against the presence of a tricuspid murmur, and a first sound that gradually gets more free from murmur on listening towards the axilla is strong evidence against the presence of a mitral murmur.

As has already been said, this tricuspid murmur does not occur in all cases of tricuspid regurgitation. The structure of the valve is such that leakage very often occurs without the production of a murmur.

SUPERIOR VENA CAVA MURMUR. — The second point at which a murmur can arise is at the place in the superior vena cava, where its walls are strengthened on its passage through the pericardium by the fibrous support given by that structure. When the vein is over-distended by the blood which is regurgitating through the tricuspid valve, this part dilates to a less extent than the rest, and a murmur is produced by the constriction thus caused.

This murmur may well be called the superior vena cava murmur.

Its characters closely resemble those of the aortic systolic murmur, and the incautious observer may very readily mistake the one for the other. The diagnostic points are, however, clearly marked if they are looked for. They are, firstly, the point of maximum loudness: this is lower in the superior vena cava murmur than in the aortic systolic. The venous murmur is usually as well heard in the third as in the second interspace, and sometimes in the fourth also, whereas the aortic murmur is not heard as low as this.

Secondly, the nature of the vessel up which the murmur is conducted. It is not always as easy to determine this as might be supposed, because of the close proximity of the veins and arteries. The character of the pulsation is often a useful guide, being weaker and more fluttering in the veins, stronger and more even in the arteries. And also, where the tricuspid regurgitation is not very extreme, the murmur and the thrill which may accompany it may often be observed to stop short suddenly in a manner suggestive of the regurgitation being arrested by competent valves in the vein.

Where the regurgitation is great and the valves are incompetent, the diagnosis is more difficult; but with care the arterial pulsation can be distinguished from the venous, and the absence of thrill and murmur in the arteries, together with their presence in the veins, will make the diagnosis clear.

Another point that often helps to differentiate these two murmurs is that the aortic murmur is as a rule better conducted along the line of the subclavian artery than the venous murmur is along the subclavian vein, owing to the presence of the venous valves.

Thirdly, the relative loudness of the murmur in the neck, as compared with its loudness in the second right interspace, may prove of service for diagnosis. In the case of the aorta, the loudness in the neck is always equal or nearly equal to its loudness over the second rib or interspace.

In the case of the murmur of tricuspid origin, its loudness in the neck depends upon the degree to which the valves in the veins have given way. If there be free regurgitation up the veins, the murmur may be as well conducted—at all events for a short distance—as the aortic murmur is. As a rule, however, this is not the case, and the conduction is not loud.

Fourthly, another important diagnostic sign is the relationship between the loudness of the first sound and the murmur as heard in the arteries of the neck. If, as in the last case referred to, the first sound of the heart can be heard over the arteries in the neck, while the murmur is heard faintly or not at all, it is strong evidence against the murmur being of aortic origin.

MURMUR PRODUCED AT THE VENOUS VALVES.—The third point at which a murmur may be caused by tricuspid regurgitation is at one or other of the valves which limit the auriculo-venous reservoir. Little need be said on this point, as it is not very common for a murmur to arise at these valves, and when such a murmur is caused, the diagnosis is not difficult, and it is not likely that any one who recognises the possibility of such a murmur occurring would be misled by it. Such a murmur may occur at the valves guarding the entrance of the external jugular into the subclavian, or those at the lower end of the internal jugular. A case of this latter nature is recorded at p. 291.

SOUNDS ARISING IN THE VENOUS VALVES.—Another physical sign of some interest, but of no great practical importance, is a sound produced in the subclavian or innominate veins by the forcible closure of their valves in cases of well-marked tricuspid regurgitation. These valve sounds are not often heard, and may cause some uncertainty when they are, because of their occurrence in the short pause of the heart shortly after the first sound and towards the end of the tricuspid systolic murmur—if there be one present. More than once, I have been able to observe that, with increasing tricuspid regurgitation, the sound of valve closure gave place to a murmur produced in the vein; or, on the contrary, as the patient improved, I have observed a late systolic murmur—which from its situation I had recognised as arising in a vein—disappear, and its place taken by the sharp sound due to the closure of the venous valves. The first time I recognised this latter phenomenon was in a case of severe aortic regurgitation with a large amount of tricuspid regurgitation. On admission to hospital there was a loud late systolic murmur audible just above the clavicle on both sides and of rather a more hissing character than cardiac murmurs usually are. In a few days the murmur on the left side of the neck was no longer audible, but the sharp sound of valve closure was heard instead, and then a few days later, the one on the right side of the neck disappeared in the same way. I have not infrequently been able to demonstrate this sound of valve closure in cases of tricuspid regurgitation.

The following cases illustrate some of the points just dealt with:—

In the following case, it was not at all easy to exclude the existence of an aortic systolic murmur in addition to the tricuspid murmur, which was evidently present. M. T., aged sixty-six, was admitted to hospital suffering from bronchitis and dyspnœa, and some œdema of the ankles. The arteries were atheromatous and the arcus senilis well marked. The apex beat was much displaced downwards and outwards, being in the sixth interspace nearly in the anterior axillary line, 5½ inches from the sternum. This was seemingly due to displacement of the heart and not hypertrophy, because there was almost no cardiac dullness near the apex beat, and the resonance of the stomach met that of the lung in, and just external to, the nipple line —i.e., between the apex beat and the rest of the cardiac dullness. Internal to the nipple line there was a little relative cardiac dullness near the left edge of the sternum, and over this dull area there was a loud systolic murmur, certainly of tricuspid origin. It was conducted upwards, and was well heard to the right of the sternum and into the vessels of the neck. It was particularly well heard over a vessel which could be felt strongly pulsating nearly half-way along each clavicle. The pulsation was strong and the vessel felt just like an artery. On careful palpation, however, the carotid could be felt internal to it, and the murmur was not heard in the carotid, and, moreover, the pulsation in the previously mentioned vessel stopped short suddenly some little distance up the vessel, in a manner very suggestive of the presence of valves checking the regurgitation up a vein.

In this case, the diagnosis of the presence or absence of aortic disease practically turned upon the question as to the nature of the vessel in which the murmur was heard, and it required a careful examination to arrive at a certainty. The extreme displacement outward of the apex beat would also, in the absence of careful percussion, have been misleading, suggesting valvular disease.

Much difficulty is also apt to arise in cases such as the following, where there is little or no systolic murmur audible over the tricuspid valve, but in which there is a well-marked systolic murmur audible in the second and third interspaces near the sternum. In such cases there is a considerable danger of mistaking this murmur for one arising at the aortic orifice, and the writer has a very distinct recollection of a systolic murmur being demonstrated in a large tutorial class at

Edinburgh in his student days as one due to aortic stenosis, which did not, to his mind, fulfil the requirements of an aortic systolic murmur, and which was certainly this superior vena cava murmur of which we are speaking.

The following is a well-marked case showing this murmur:

The patient was a man aged sixty-two, who was admitted to the Birmingham General Hospital some years ago, under my care, suffering from shortness of breath and dropsy. He had well-marked atheroma of his arteries, and there was a well-marked systolic murmur audible over the second right interspace close to the sternum, and conducted upwards into the neck. This murmur might easily be mistaken for one of aortic origin. The following characters showed that it was not. The murmur was more loudly heard over the second right interspace than it was in the neck. This, as has been pointed out, was against it being of aortic origin.

Again, on comparing the relative loudness of the first sound and the murmur it was quite clear that the first sound of the heart was more perfectly conducted along the arteries of the neck than the murmur was. This would not be the case if the murmur were being conducted along the arteries. If both the murmur and the first sound were being conducted along the same channel the loudness of the two sounds ought to bear a constant relation to each other, and one would not vary materially without the other. This fact that the first sound of the heart was conducted along the carotids more perfectly than the murmur, was conclusive evidence to my mind against the aortic origin of the murmur.

In spite, therefore, of the inherent probability, in view of the atheroma of his arteries, that the murmur was due to roughening of the aortic orifice, I was compelled to diagnose the murmur as being due to tricuspid regurgitation, and this also in spite of the fact that there was no murmur audible over the tricuspid valve or over the right ventricle.

After a few days' rest in bed and tonic treatment, the œdema subsided and the murmur became variable—as was to be expected considering its tricuspid origin—and soon entirely disappeared and thus justified what appeared to some of those who examined the patient, a 'foolishly fanciful' diagnosis, considering the reasonable probability of an aortic murmur being present. On referring to the notes of the case, I found that my then house physician, relying on the orthodox description of the aortic systolic murmur, preferred to enter up the case in the records as one of 'aortic disease.'

Considering the difference, both as to prognosis and treatment, between aortic disease and tricuspid regurgitation, the importance of clearly recognising the points of diagnostic value is evident, more especially as the diagnosis is often not easy—as the last case I have quoted testifies.

The signs of diagnostic value to which I would draw attention are as follows:—

(*a*) The character of the murmur.—It is apt to be softer and more blowing in tricuspid regurgitation, though not invariably so.

(*b*) The nature of the vessel up which the murmur is conducted.—It is not always as easy to determine this as might be supposed, because of the close proximity of the veins and arteries. The character of the pulsation is often a useful guide, being weaker and more fluttering in the veins, stronger and more even in the arteries. Also the pulsating parts of the vessel can often be felt to come to an end suddenly in the case of a vein, owing to the regurgitation being arrested by a competent valve.

(*c*) The relative loudness of the murmur in the neck as compared with its loudness in the second right interspace may prove of service for diagnosis. In the case of the aorta, the loudness in the neck is always equal or nearly equal to its loudness over the second rib or interspace.

In the case of the murmur of tricuspid origin, its loudness in the neck depends upon the degree to which the valves in the veins have given way. If there be free regurgitation up the veins, the murmur may be as well conducted—at all events for a short distance—as the aortic murmur is. As a rule, however, this is not the case, and the conduction is not loud.

(*d*) Another important diagnostic sign is the relationship between the loudness of the first sound and the murmur as heard in the arteries of the neck. If, as in the last case referred to, the first sound of the heart can be heard over the arteries in the neck, while the murmur is heard faintly or not at all, it is strong evidence against the murmur being of aortic origin.

The following case of a superior vena cava murmur is of sufficient interest to make it worth recording here.

The patient was a boy aged fourteen, who was suffering from a rapidly developing attack of pneumonia with the usual adolescent type of upward dilatation of the right ventricle.

There was only half an inch or so of relative dullness in the second left interspace; but in the third space there was relative dullness and pulsation to the nipple line (about 2 inches from the sternum), and in the fourth to the anterior axillary line 3½ inches from the sternum. The diaphragm was high on the left side, for there was full gastric resonance in the fifth interspace for nearly 3 inches; but beyond this point there was cardiac pulsation due to the apex beat, which was palpable 3½ inches from the lateral sternal line. On the right side the diaphragm—as judged by the level of the liver dullness—was normal (*vide* fig. 58).

Systolic Murmur Arising in the Superior Vena Cava

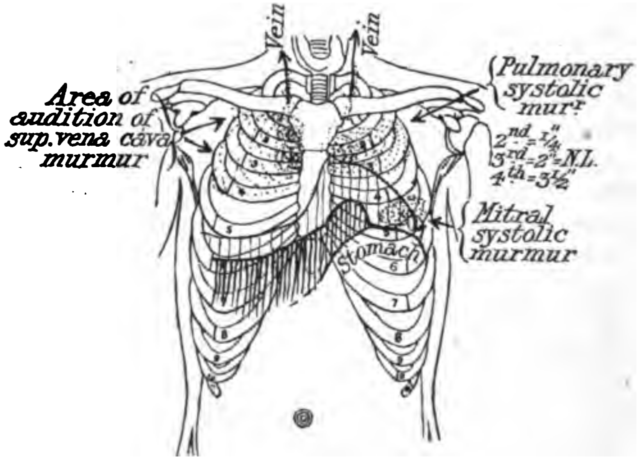


Fig. 58.

Thomas W., aged fourteen. Acute pneumonia. Loud pulmonary and superior vena cava murmurs. High gastric resonance. Faint mitral systolic, pulmonary second sound *not* loud. Veins of neck *not* full. Area of audition of murmurs shown by dotted shading.

There was no increase of the cardiac dullness to the right, and no excess in the amount of right ventricular pulsation palpable to the left of the sternum, and there was no tricuspid systolic murmur; the first sound was clear and sharp over the lower end of the sternum and over the liver.

The pulmonary second sound was not accentuated and was, if anything, less loud than normal. Also there was no over-fullness of the neck veins.

In spite, however, of all these signs that the right ventricle was neither distinctly dilated to the right not developing more force than normal, there was a very loud murmur which, from its point of maximal loudness and its direction of conduction, must have been produced in the superior vena cava and caused by tricuspid regurgitation.

It had its point of maximal loudness in the third and second right interspaces, and was heard well over a considerable part of the right pectoral region to beyond the right nipple line, and loudly conducted into the veins of the neck.

This case is not very easy of explanation.

The presence of a high diaphragm in a case of acute pneumonia is suggestive of muscular weakness of the heart, as is also the fact that the only sign of increased right ventricular action was a slight increase in the loudness of the right ventricular first sound, although the ventricle must have been embarrassed not only by the pneumonia, but also by the failure of the left ventricle, which was shown by its dilatation and the presence of a faint mitral systolic murmur.

The outward dilatation in the third left interspace and a well-marked pulmonary systolic murmur also pointed to overstrain of the right side of the heart.

The reason for the absence of more signs of venous engorement must be sought for in the supposition that the muscular tone of the veins was good, and the reason for the loudness of the superior vena cava murmur in the supposition that for some reason or other (possibly dependent on the great dilatation to the left and on the presence of the pneumonia), the superior vena cava was more constricted than it usually is at the point where it passes through the pericardium.

Arterial Compression Murmur

In connection with the diagnosis of systolic murmurs, audible along the line of the vessels, it is well to remember the possibility of a systolic murmur being produced in the subclavian artery itself. This murmur, which is audible in the region of the clavicles, may, without proper care, be mistaken for a systolic pulmonary one or else, if well marked on the right side, for a murmur due to tricuspid regurgitation. In character it is a sharp blowing systolic murmur audible more or less widely, whose point of maximal loudness lies at about the spot where the subclavian artery crosses the first rib. The murmur often closely resembles a loud pulmonary or aortic systolic murmur or a loud superior vena cava murmur, but it can be at once distinguished by two characteristics: firstly, the situation of its point of maximal intensity; and secondly, the fact that it can be caused to disappear by altering the position of the shoulder.

There seems no doubt that it is produced in the subclavian artery, at the point where it crosses the first rib, and it probably is caused by compression of a badly filled artery at this point.

Without due care this murmur is quite apt to cause an error in diagnosis.

It is specially common in young women with anæmia.

A similar murmur can often be easily produced by too firm pressure of the stethoscope over the subclavian artery below the clavicle.

PART II.—ESSAYS DEALING MAINLY WITH DILATATION OF THE HEART DUE TO OVERSTRAIN

ESSAY V.—THE CONDITION OF THE HEART IN THE ANÆMIA OF ADOLESCENCE

THE cases of anæmia studied in this research were almost entirely the ordinary cases of chlorosis and anæmia which occur so frequently when young women of eighteen to twenty-two engage in manual work under the unsatisfactory conditions which used often to be associated with work in factories and workshops. I have excluded cases where the anæmia was definitely secondary to such diseases as phthisis and chronic nephritis, because the cardiac phenomena are usually different from those which occur in the simple anæmia of adolescence. In the cases dealt with in this essay, the anæmia was a purely functional one, and subsided on the adoption of treatment by rest and iron.

AS REGARDS THE METHOD OF EXAMINATION

The patients were as a rule examined in the recumbent attitude and often when erect as well. In connection with the somewhat extreme amount of dilatation which is often noted, the following facts must be remembered :—

1. This research deals with a consecutive series of cases observed twenty years ago when the conditions of factory labour were more calculated to produce anæmia than they are at the present day.

2. The heart was often percussed with the lungs as fully deflated as the patient was able to manage—so as to get as nearly as possible the absolute size of the heart.

3. As the patients were not examined oftener than once a week, and always improved under treatment, it follows that the greatest amount of cardiac dilatation was found on first examining the patient. Now the subjects of this type of anæmia are mainly young women from seventeen to twenty-one years of age, in whom a careful examination of the chest at their first visit to a hospital is apt to be accompanied by a certain amount of nervousness. This nervous excitement would naturally tend to exaggerate, temporarily, such cardiac dilatation as was already present. Even though such temporary exaggeration of the amount of dilatation were present, however, it does not in the least detract from the value of the symptoms presented or the evidence they afford as to the changes which take place when the heart fails, in consequence of anæmic malnutrition. From this research, I have excluded all cases where the failure of the right ventricle was secondary to organic valvular disease of the heart.

THE CONDITION OF THE HEART IN THE ANÆMIA OF ADOLESCENCE

The careful clinical study of the heart in anæmia, which this research entailed, demonstrated quite clearly that the most important change, which occurs where heart failure accompanies the anæmia of adolescence, is a dilatation of the right ventricle.

EVIDENCES OF DILATATION OF THE RIGHT VENTRICLE

Inspection and palpation in cases of anæmic dyspnœa in adolescents give the physical signs of enlargement of the heart, which have been already described (pp. 14, 25) as characterising dilatation of the conus arteriosus of the right ventricle.

DILATATION UPWARDS AND TO THE LEFT

Percussion of the heart in cases of anæmic heart failure reveals the fact that there is an enlargement of the heart in the third and second left interspaces in the great majority of those who are suffering from dyspnœa due to this cause.

The following statistics bearing upon this point were gathered in my out-patient department at the Birmingham General Hospital in the twenty-one months between September 1890 and May 1892. During this period, 350 cases of this type of anæmia were seen. In 63 cases no notes were taken, and only the remaining 287 are therefore available for study.

Out of the 287 cases of anæmia, the heart was normal in 45, and showed some signs of failure in 242.

The presence or absence of dilatation of the heart upwards and to the left in the second and third left interspaces was noted in 220 cases out of the 242, and the results will best be stated in tabular form.

CASES OF SIMPLE ANÆMIA SEEN BETWEEN SEPTEMBER 1890 AND MAY 1892

No detailed notes taken	63	cases
Heart normal	45	,,
Some cardiac failure, but character not noted .	22	,,
Dyspnœa, but no dilatation in second interspace	35	,,
Upward dilatation of the right ventricle 'present,' only 'very slight,' or 'amount not noted'	18	,,
Amount of dilatation carefully noted . .	167	,,
	350	,,

With regard to the 167 carefully noted cases—

In 73 of them the amount of relative cardiac dullness in the second left interspace was 1 inch or less in extent (in 35 it was 1 inch in extent).

In 71 patients the relative dullness in the second left interspace was more than 1 inch in width. Out of these 71 cases there was more than 1½ inches of dullness in 27 patients.

In 23 patients, although there was no cardiac dullness in the second interspace, there was a distinct increase of the dullness in the third left interspace.

The exact amounts of relative dullness in the second left interspace in the 167 patients were as follows:—

In 19 patients there was ½ inch of relative dullness.
„ 19 „ „ „ ¾ „ „ „ „
„ 35 „ „ „ 1 „ „ „ „
—
73
—

In 17 patients there was 1¼ inches of relative dullness.
„ 27 „ „ „ 1½ „ „ „ „
„ 7 „ „ „ 1¾ „ „ „ „
„ 14 „ „ „ 2 „ „ „ „
„ 3 „ more than 2 „ „ „ „
„ 3 „ there was 3 „ or more „ „
—
71
—

In 23 patients there was no relative dullness in the second left interspace, but there was increase of relative dullness in the third interspace—1½ inches being taken as the normal amount.

In 14 patients there was 2 inches of relative dullness.
„ 5 „ „ „ 2½ „ „ „ „
„ 2 „ „ „ 3 „ „ „ „
„ 2 „ „ „ 'some dilatation'
—
23
—

In many cases, where there was more than one inch of relative dullness in the second left interspace there was pulsation to be seen and felt. In some there was absolute dullness as well as relative (*vide* fig. 77, p. 268).

It must therefore be acknowledged that increase of the cardiac dullness upwards and to the left is very frequent in cases of anæmia with heart failure, for it occurred in 185 out of 350 cases—that is, in 54 per cent.

If, however, the cases showing signs of heart failure alone are considered, the frequency of this type of dilatation is found to be at least 84 per cent.

It can therefore be definitely asserted that increase of the heart upwards and to the left in the second and third left interspaces is usually found, whenever marked dyspnœa accompanies the simple anæmia of adolescence.

THE CAUSE OF THE UPWARD INCREASE OF THE HEART

The question must now be answered: To what is this increase in the cardiac dullness in the second left interspace due? It might be due to the left auricle, the pulmonary artery, or to an upward increase of the right ventricle.

THIS INCREASE IN THE CARDIAC DULLNESS IS NOT DUE TO THE LEFT AURICLE

In all cases, where the cardiac dullness in the second left interspace is well marked, there is, as a rule, visible pulsation as well; therefore it is quite easy to determine the nature of this pulsation, if the cardiac sounds be followed by the stethoscope at the same time that its rhythm be watched. When this is done, the pulsation is found to be certainly systolic and not pre-systolic in rhythm. I have studied this rhythm in many hundreds of cases, and in no more than one or two, at most, have I failed to satisfy myself of its systolic nature. On first commencing this research, I was under the impression that the pulsation was due to the auricle, as brief notes made of my out-patients at the hospital in 1889 and 1890 testify; and, therefore, the conviction that it is always systolic (at which I very speedily arrived) is the result of observation and not of theory. Cardiographic observations—of which I have taken some hundreds—have also demonstrated this same fact.

It is, however, physically possible for a distended left auricular appendix to reach the chest wall in the second left interspace, as may be demonstrated if the left side of the heart be distended *in situ* by hard paraffin, when the right side is empty. There is, however, good reason to doubt if this ever actually occurs clinically, for the distended right side of the heart will prevent the appendix from reaching the anterior chest wall.

INCREASE OF CARDIAC DULLNESS NOT DUE TO THE PULMONARY ARTERY.

That it is not due to the pulmonary artery is shown by the use of the cardiograph, for the auricular wave is shown in a manner that would be impossible in the case of the pulmonary artery. Also by carefully timing the pulsation, the maximum rise can, not infrequently, be clearly seen to be systolic in rhythm, and to subside considerably, before the second sound of the heart, in a way that could not occur if it were due to the pulmonary artery.

Further, in well-marked cases, the shock due to closure of the semilunar valves can, sometimes, be clearly felt at the upper level of the dull area (p. 286). In no case have I been able to find any evidence that it was due to the pulmonary artery. That it is due to one of the ventricles is shown by the above observations.

INCREASE OF CARDIAC DULLNESS DUE TO RIGHT VENTRICLE

That it is due to the right ventricle is shown, firstly, by the fact that the amount of the pulsation, observable over the dull area, varies with respiration. It is most marked at the end of expiration. That this is not due to the recession of the lungs, during expiration, is shown by the fact that it is not so well marked when the chest is held in the position of full expiration, as it is during ordinary breathing.

It cannot be believed that the left ventricle could be influenced to this extent by the respiratory movements. But, considering the relatively low pressure in the right ventricle, it is, I think, to be expected that the variation in the intrathoracic pressure, which results from the respiratory movements, would influence the filling of the heart sufficiently to cause the above-mentioned variation.[1]

---

[1] Numerous observations made by the writer in recent years on the heart sounds prove that the sounds due to the right ventricle are very frequently louder at the end of inspiration than at any other time in the respiratory cycle, the same variation not being observable in the case of the left ventricle.

Pathological Proof.—I have seen many post-mortems on well-marked cases of anæmia, and have frequently distended the heart with hard paraffin *in situ* in such cases, and the pathological evidence is clear that the upward enlargement of the heart is due to dilatation of the right ventricle.

The following are the notes with regard to the relation of the undistended heart to the chest wall in one such case, and of the distended heart in another case :—

Kate G., aged fifteen. Extremely anæmic. Heart extremely dilated, with well-marked pulmonary murmur.

The relative dullness of the heart during life was as follows :—

About 1¼ inches in second left interspace.
„ 2½ „ „ third „ „
„ ½ inch „ third right „
„ 1 „ „ fourth „ „
Apex fifth left space in the nipple line.

The girl died, suddenly, three days after the above notes were made.

The following notes were made as to the position of the heart in relation to the chest wall at the post-mortem examination :—

Right ventricle.—The conus arteriosus is much dilated upwards, and reaches from the right edge of the sternum in the first space to the lower border of the first rib on the left, and extends for 1½ inches in the second left space.

The pulmonary valves were under the lower border of the first rib ; the pulmonary artery in its collapsed condition looked crumpled and irregular, and the dilated upper border of the conus arteriosus overhung it.

In another case, an equal degree of dilatation of the upper part of the right ventricle was observable. The following are the details of this case :—

The patient was a young man suffering from a rapidly progressing pernicious anæmia.

In this case also, the conus arteriosus of the right ventricle extended from the second right costal cartilage upwards to the lower part of the first rib, and outwards in the second left space as far as the nipple line.

It is needless to say that here, as in the last case, the left auricular appendix in its relaxed condition was completely hidden behind the dilated right ventricle. The right auricular appendix came up as high as the first right space, and its tip reached to the middle line of the sternum.

The apex was in the fourth space, one inch external to the nipple line.

This is, in fact, the type of dilatation already fully described at pp. 12 and 25, and also dealt with at p. 330, and is the usual type that occurs in the right ventricular failure in valvular disease in puberty and adolescence.

In such cases, this type of dilatation is often extreme in amount, and it is not very uncommon to find—if the heart be distended *in situ* with hard paraffin—at the post-mortem, that there is so much upward dilatation of the conus arteriosus of the right ventricle that the pulmonary valves lie in the upper part of the second left interspace or even under the first rib.

For further proof, see also p. 247.

CONSEQUENCES OF THIS TYPE OF DILATATION OF THE RIGHT VENTRICLE.—The most important change resulting from this dilatation is an alteration in the relationships of the pulmonary artery, whereby a murmur is apt to be produced with each systole of the ventricle. The further discussion of this point will be taken up later on, when the cardiac murmurs which occur in anæmia are considered.

DISPLACEMENT OF THE APEX UPWARDS.—Another striking change, which is noticeable in cases of heart failure in anæmia, is the upward displacement of the apex. This change is described by the late Dr. Foxwell,[1] and the conclusions at which I have arrived, as the result of my observations, are, in the main, identical with his. This upward displacement of the apex is common in this type of heart failure. Thus out of a total of 192 cases, where there was upward dilatation of the right ventricle, the apex was above the level of the fifth interspace in 87—i.e., in 45·3 per cent.

---

[1] *Essays in Heart and Lung Disease, vide* p. 238.

In one-quarter of these 87 cases the elevation was not very great, and the apex was described as being under the fifth rib. In some of these last-named cases the apex was palpable in the fourth interspace when the patient was recumbent, but could be felt in the upper part of the fifth interspace, when the patient was erect.

It is safe to say that in nearly half the cases of well-marked upward dilatation of the right ventricle in anæmia, there is upward displacement of the apex.

The following are some cases which illustrate this upward displacement of the apex.

Firstly, an extreme case is the following:—

A young woman suffering from anæmia and dyspnœa had so great an amount of dilatation of the conus arteriosus and adjacent part of the ventricle, combined with upward displacement of the apex, that there was well-marked cardiac pulsation for more than 2 inches in the second left interspace, while the main cardiac impulse was in the third interspace, and there was no cardiac pulsation in the fourth interspace, except during inspiration, when the elevation of the ribs allowed of the apex being felt in the upper part of that interspace. Cardiograms taken in the third interspace showed the pulsation there to be of the true apex type (*vide* p. 298).

The following case, although not one of anæmia, is of exceptional interest, because it throws some light upon the cause of the changes in the heart, which we are now discussing. For permission to refer to it, I am indebted to my colleague Sir Robert Simon, under whose care the patient was, while an in-patient in the Birmingham General Hospital.

Case showing displacement of apex. The girl, aged about fifteen, had suffered from occasional fainting attacks for some years before I first saw her in October 1891, and after each fainting attack, she suffered from dyspnœa, which had increased latterly. During the last few months, cyanosis had developed. When I saw her, the heart presented a condition similar to that above described. There was, however, no hæmic murmur in the second space, and there was cyanosis instead of anæmia.

She shortly after developed thrombosis of the right subclavian vein, and was admitted to the hospital, where she remained till death, four months later. The thrombosis subsided, but her cardiac condition remained unaltered, and she died of heart failure. The post-mortem showed no organic disease of any sort. The right side of the heart was dilated, the left side rather smaller than normal, and the aorta was very much narrowed. The narrowing was at first thought to be congenital, but I now believe it to be simply due to the lessened work thrown on the left ventricle.

Some idea of the dilatation of the right ventricle will be given by the following figures:—

The total circumference of the heart was 11½ inches. Of this, 7 inches belonged to the right and 4½ inches to the left ventricle. The left ventricle was situated wholly posteriorly, and the right ventricle occupied the whole of the front and sides of the heart.

As to the cubic contents of the chambers (measured by distensible india-rubber bags under a pressure of about 2 feet of water) it was as follows:—

Left ventricle, 90 c.c. (i.e. about normal).
Left auricle, 35 c.c. (normal, 90 c.c. more or less).
Right ventricle, 160 c.c.
Right auricle, 215 c.c.

The right auricle only projected 1½ inches to right of the middle line. The tip of its appendix was under the sternal end of the second left rib. The auriculo-ventricular groove was wholly to the left of the sternum, and it commenced above at the lateral sternal line, under the second rib, and ran downwards and slightly outwards to the level of the fifth costal cartilage at ½ inch to the left of the lateral sternal line. This point was as low as, if not lower than, the level of the apex, which was situated in the upper part of the fifth space in the anterior axillary line.

The nature of this case is extremely obscure, and I can only explain it by supposing some hindrance to the passage of the blood through the lungs. Nothing definite was, however, found that would clear up this point.

The interesting point about this case is, that we have here a case of dilated and hypertrophied right ventricle uncomplicated by other disease, and it presents just the clinical appearances of the dilated heart of anæmia, and helps to elucidate the question we are now discussing as to the displacement of the apex beat.

In the present instance, the axis of the heart seemed much more horizontal than usual, and the position and direction of the auriculo-ventricular groove was altered. It was wholly displaced to the left (probably by the distension of the right auricle), and its lower end was also swung to the left.

This rotation would, of course, tend to raise the apex somewhat, and its influence would be added to the other agencies at work.

As regards the cause of this displacement of the apex:—

1. Some part of the displacement may be only apparent, due to the distension of the right ventricle preventing the true apex from striking or even coming near to the chest wall.

2. A part of the displacement may sometimes be due (as seemed to be the case in the patient referred to on p. 353) to the distension of the ventricle downwards. I think this is improbable in most cases of anæmia, because we rarely find any dilatation of the right auricle.

3. A rise in the average level of the diaphragm does, undoubtedly, play some part in causing a rise in the heart's apex in some cases of anæmia, but in the statistics here quoted this point had not been recognised and was therefore not inquired into.

It is certain that a high apex beat is often seen in anæmia without any corresponding rise in the diaphragm. The whole question of the exact relationship which a rise in the average level of the diaphragm bears to anæmia is not one upon which I can, at present, speak definitely.

4. It seems clear that the rise of the apex is due to a dilatation of the main portion of the anterior wall of the right ventricle. This increase does not take place to the right—as might be expected; nor forwards, owing to the support given by the anterior chest wall; nor backwards, because of the strength of the interventricular septum, and therefore has to take place to the left. To do so, the left ventricle has to move outwards, and can only do so by swinging round its fixed point at the base. In so doing, the apex must move in the arc of a circle, whose centre is at the root of the aorta; and the apex must therefore move upwards, as well as outwards, in order

to give room for the expanding right ventricle, as was pointed out by the late Dr. Foxwell in his 'Essays in Heart and Lung Disease.'

Reference to the figures, showing the shape of the heart in anæmic dyspnœa (which are given on pp. 261, 268, 289, &c.), illustrate this point as to the upward displacement of the apex. The discussion of the various factors at work in producing this displacement is fully dealt with in the essay on displacement of the heart.

GENERAL DILATATION TO THE RIGHT IN ANÆMIA

Another point of interest in connection with this type of cardiac dilatation is the absence of any enlargement of the heart towards the right.

Taking the relative cardiac dullness to extend normally for half an inch to the right of the sternum (if the chest is percussed in the position of full expiration), and reckoning as abnormal any amount of relative dullness which exceeds this half-inch, we find that among the 178 cases of well-marked anæmic dilatation of the heart only 15, or not quite 8·5 per cent. showed dilatation of the heart to the right—i.e., the right border of the heart was normal in 91·5 per cent. of the cases.

The relative cardiac dullness in anæmia frequently does not reach as far as this (which is given as the normal limit of the relative cardiac dullness to the right); but the same can be said as regards the percussion of any normal chest.

The further details as to the situation of the right border of the cardiac dullness in these cases of anæmia are as follows: right border, ½ inch from sternum, 23 cases; ¼ inch, 9 cases; at right edge of sternum or mid-sternum, 42 cases, and no definite note in 89 instances. Among the 15 cases showing dilatation to the right of the sternum, there were only 6 where there was 1 inch or more of relative dullness on percussion.

In the face of such figures as these, it can be asserted that there is, as a rule, no dilatation of the heart to the right in cases of simple anæmia in adolescence showing upward dilatation of the right ventricle.

This fact is strong evidence in favour of the statements advanced in an earlier essay as to the reason why this type of dilatation occurs in adolescence.

It is more than likely that this absence of dilatation to the right is not a mere coincidence, but that the two facts stand in the relationship of cause and effect.

The upward dilatation of the right ventricle is caused by the intraventricular pressure being in excess of the resisting power of this the weakest portion of its muscular wall, and the greater the intraventricular pressure the greater will be the dilatation. For the development of this pressure it is essential that the tricuspid valve should be competent, otherwise the 'safety valve' action of the heart would come into play and tricuspid leakage prevent the intraventricular pressure from rising to an adequate extent. In the adolescent the softness of the fibrous tissues, owing to their immaturity, will probably render the right auricle and veins less able to give support to a leaking tricuspid valve than is the case with the stronger fibrous tissues of the adult. Certain it is that dilatation of the right ventricle to the right and the tricuspid incompetence to which it leads are very rarely found to be present in the heart failure with upward dilatation of the right ventricle which occurs in the anæmia of adolescence.

Frequency of a Tricuspid Systolic Murmur

Evidence as to the infrequence of general dilatation is also given by noting the occurrence of a tricuspid systolic murmur in cases of heart failure in anæmia. Out of the 178 cases, the presence of a murmur was only noted in 41 instances, or 23 per cent. of the cases, and of these the murmur was a faint one in 12 instances; so it was well-marked in only 29 patients—i.e. in 16 per cent. If the 178 cases of anæmia be divided into two groups according to age, it will be found that in the older group the number of cases of tricuspid regurgitation predominate, thus confirming the statement made (p. 33) that the older the patient the greater the tendency to general dilatation of the right ventricle rather than to the localised dilatation of the conus arteriosus and the adjacent part of the anterior wall which occurs in adolescence. The figures are as follows: In cases of eighteen years of age or less, a tricuspid murmur was present in 15 per cent., whereas in patients of nineteen years or more it was present in 28 per cent. of the cases.

Out of the 41 patients just referred to who had a tricuspid systolic murmur, only 8 gave any evidence of dilatation of the heart towards the right, and in the remaining 33 the right border of the heart was normal. This leaves 8 cases in whom there was some dilatation to the right without any evidence of a tricuspid murmur. For illustrations of the area over which the murmurs were audible in the above cases of anæmia, see figs. 75, 78, 81, and 86–90.

The total number of the 178 patients who showed some signs of general dilatation of the right ventricle was therefore 47, or nearly 27 per cent.

Presence of Signs of Failure of the Left Ventricle in the Anæmia of Adolescence

In cases of anæmic heart failure, all evidence seems to show that failure of the left ventricle is not ordinarily present, although it may sometimes occur. Considering the weakness of the right side and the amount of hard work that some of these anæmic girls do in factories and elsewhere, it is interesting that left ventricular failure is not more often seen. The reason for this is to be found in the weakness of the right ventricle. Failure of the right side of the heart and the breathlessness which it causes, prevents the patient from exerting herself sufficiently to overstrain the left heart.

The full statistics of left ventricular failure are not easy to obtain, owing to the frequency with which, in these cases, the apex is swung upwards and outwards to a point in the fourth interspace external to the nipple line.

It is evident that in such cases the outward displacement of the apex is no criterion as to the presence of dilatation of the left ventricle. It is more than probable that this upward and outward displacement of the apex is evidence that the left ventricle is at most of normal size and quite possibly that it is smaller than normal.

This question is further discussed at p. 348.

If those cases only are considered, where the apex is in the fifth interspace (and they number only 93 out of the 178), it is found that 22, or 23·6 per cent., had some outward displacement of the apex, suggesting left ventricular dilatation. Of these, only 9 had a systolic mitral murmur, and 13 had not.

If the presence of a mitral systolic murmur be inquired into, it is found that 38 patients out of the 178 had this murmur present, or 21 per cent.

Taking these sets of figures together, it shows that 52 patients out of the 178, or 29·2 per cent., showed some signs of left ventricular failure.

In connection with what has been said as to the weakness of the thinner part of the right ventricle in adolescence, it is interesting to find that on noting the ages of the cases showing signs of left ventricular failure, it is found that they mainly occur amongst the older adolescents. The figures are as follows :—

On dividing the patients into two age-groups, one of eighteen and under and the other of nineteen and over, it is found that only 11 of the cases with a mitral systolic murmur, or 15 per cent., occurred in the younger group, whereas of the 107 patients in the older group, 27, or 25·2 per cent., showed the presence of a mitral systolic murmur. On analysis of the thirteen cases where the apex beat was outside the nipple line, but no mitral systolic murmur was present, it appears that seven of them were in the younger group and 6 in the older; but considering the possibility of outward rotation of the apex occurring without any actual dilatation of the left ventricle in these cases, it is preferable to rely on the presence of a murmur rather than on the position of the apex alone, as a sign of left ventricular failure.

CAUSE OF RIGHT VENTRICULAR DILATATION IN ANÆMIA

In the next place the cause of these changes in the shape and size of the heart must be dealt with.

Is it secondary to failure of the left side of the heart?

The first question to be asked is whether in anæmia the cause of dilatation of the right ventricle is the same as it is in valvular disease and most cases of overstrain, where its failure is secondary to that of the left ventricle.

Some light upon this question can be thrown by noting the frequency with which signs of left ventricular failure are noticeable in cases of anæmia, although, as already pointed out (p. 47), these signs do not become evident clinically unless well marked.

Still, if the failure we are now speaking of were secondary to failure of the left side, we should expect to find that every severe case of failure of the right ventricle showed well-marked signs of failure of the left side.

This is, however, not the case, as the following statistics show; although there is, as might be expected, more frequently a mitral systolic murmur in the severe than in the slight cases of dilatation.

Out of 213 of the more severe cases dealt with, an apical systolic murmur was only noted in 31 instances, and in 6 of these it was only a faint murmur. In only 3 cases is the murmur noted as being loud enough to be conducted to the axilla.

There can, however, be no doubt as to the important part which a failing left side plays as a rule in the causation of right ventricular dilatation, although in the cases we are now dealing with the part it plays appears to be only a very minor one.

It is possible that this type of dilatation is secondary to a strong rather than to a weak left ventricle, because a strong left ventricle will admit of far more physical exertion than would be possible if the left ventricle were weak.

The young woman whose systemic circulation is good will work till she is stopped by the breathlessness which results from the failure of her right ventricle. The patient with a weaker left ventricle will not be able to put so much strain upon her right heart (see illustrative cases, pp. 294 and 304).

Secondly, Is the failure of the right ventricle due to the malnutrition of the heart muscle, which results from the anæmia?

The answer to this question must be in the affirmative, for muscular malnutrition must be acknowledged to play some part in causing the dilatation we are dealing with.

As has already been pointed out (p. 10), in adolescence the heart depends more upon its muscular than upon its fibrous tissues for its power of resisting over-distension, and, therefore, any weakening of the power of its muscles by malnutrition must tend to increase its liability to dilatation in the face of any undue strain.

A weakly lad who over-exerts himself is more apt to suffer from cardiac dilatation than a stronger one is.

That malnutrition of muscular tissues does occur in anæmia from lack of oxygen, and possibly also of other nutritive materials carried by the blood, is sufficiently self-evident not to need further comment here.

On the other hand, it is interesting to note the difference that exists between the type of anæmia we are here discussing and the symptomatic anæmia which is secondary to some exhausting disease such as phthisis or nephritis. In such cases it seems as if the debility which accompanies these conditions so far weakens the heart that it does not develop enough interventricular pressure to cause marked dilatation.

This is clinically so certain that when in a young woman a considerable amount of anæmia is unaccompanied by any upward dilatation of the right ventricle, it is safe to presume that the anæmia is secondary to some organic disorder and is not a simple anæmia which will be curable by rest and the administration of iron.

This leads up to the next point to be considered—namely, that this type of right ventricular dilatation is not due to muscular malnutrition alone, but that another factor must be recognised as well—namely, 'overstrain.'

Thirdly, Overstrain of the right side as a factor in causing dilatation of the right ventricle in anæmia.

The clinical evidence is clear that overstrain of the right ventricle is a cause of its dilatation in the class of case of which we are now speaking.

Supposing that muscular weakness of the thinnest part of the muscular wall (owing to malnutrition) were the sole cause of the dilatation, the factors causing the over-distension would be an intraventricular pressure rather less than normal *plus* a wall too weak to withstand it. In such a case, although dilatation would occur, the work done by the muscular contraction would not be in excess of the normal, and therefore the heart sounds would not be louder than normal. Cases of this nature are sometimes met with in anæmia.

In the case of ordinary anæmic dilatation, on the other hand, the heart sounds *are* usually markedly in excess of the normal, so far as their loudness is concerned, and also the character of the visible and palpable pulsation of the right ventricle clearly indicates that it *is* doing more work than it does under normal circumstances. Measurement of the loudness of the second sound produced by the closure of the pulmonary valves shows clearly that the pressure closing them is far above the average, for the loudness of the sound is sometimes double what it is in health; for if the normal loudness of the pulmonary second sound be represented by the figures 16 to 20 or so, in anæmic dilatation of the right ventricle the sound frequently measures as much as 30 or 40, and sometimes as much as 48 (see pp. 415 and 420, 'Theory of Compensation').

Further, in these cases the sound produced by the closure of the pulmonary valves can often be heard distinctly to precede that produced by the aortic valves, causing a reduplication of the second sound, thus proving that the tension in the pulmonary artery is in excess of the normal, and therefore causing the valves to close earlier than usual.

Both of these observations show that in anæmic dilatation of the heart the right ventricle is developing more force than normal, and that therefore overstrain must be acknowledged as a factor in the causation of the dilatation. As already pointed out this overwork might be suspected to be due to the interference with the pulmonary circulation, which a failing left ventricle would bring about were it not that, in the majority of the cases, there is no evidence at all that there is any such failure.

Moreover, the careful measurement of the loudness of the pulmonary second sound in cases of valvular disease, where the left ventricle *is* failing, shows that its loudness in such cases does not reach the extreme degrees which it does in cases of anæmic dilatation of the heart.

For in cases of failing compensation in valvular disease the loudness of the pulmonary second sound would only be represented by the figures 9 to 24, and very rarely as much as 28 or 30 in comparison with the figures of 30 and 40, and even 48, which are obtained in anæmia.

These observations show that there is more cause for embarrassment of the right ventricle in cases of anæmia, than the overstrain which results from failure of the left ventricle when there is no anæmia.

Therefore, the statement that overstrain is a factor in the causation of this type of dilatation of the right ventricle can not, only be confirmed, but also exemplified as follows :—

The dilatation of the right ventricle, which occurs in cases of anæmia, is due in part to the increased work which is thrown upon the right ventricle by some interference with the circulation of the blood through the lungs, which is consequent upon the abnormal condition of the blood.

While observations on the condition of the heart and circulation in cases of anæmia show the presence of some hindrance to the passage of the anæmic blood through the lungs, there is no evidence at present to show upon what the cause of the embarrassment depends.

The natural supposition would be that the failure in the oxygen-carrying power causes a contraction of the pulmonary venules, so as to keep the blood in contact with the air in the lungs for as long a time as possible to enable the blood to take up the maximum of oxygen which it is capable of holding ; but against such a supposition must be urged the fact that physiologists tell us that they know of the existence of no such regulating mechanism in the pulmonary circulation.

Nevertheless, it is possible—in this case as in some other vital phenomena—that the physiologist must learn from the clinician, instead of the physiologist showing the way, as he usually does.

Another alternative is that there may be an increase in the total volume of the blood in these anæmic states, and that in this way extra work is thrown upon the right ventricle; and the researches made in this direction by the carbon monoxide method would, if confirmed, give some weight to the suggestion. This cannot, however, explain all cases, for it is quite certain that in many cases of anæmia the clinical evidence points conclusively to the total volume of the blood in active circulation being diminished and not increased.

It is quite evident that further research, both clinical and experimental, is required before this question can be decided; and most interesting results would be obtainable from a long series of cases of anæmia, where the exact loudness of the pulmonary second sound was carefully noted as well as the loudness of the pulmonary murmur, the amount of cardiac dilatation, and the level of the diaphragm.

I regret that in my series of cases the loudness of the pulmonary second sound and the level of the diaphgram were not accurately noted, and therefore I am unable to speak definitely upon the varying degree of resistance to the pulmonary circulation, which was present in them. Neither have I the opportunity of collecting another group of cases, because observations such as these upon the relative loudness of the heart sounds cannot be spread over several years, owing to the variation in the 'personal equation' which is unavoidable in our present method of measuring the loudness of the cardiac sounds. Such observations, to be fully reliable, ought to be made upon a continuous series of cases seen within a short time—conditions which are afforded by hospital out-patient practice in a manufacturing town. When, however, the 'relay telephone' can be adapted to the recording mechanically of the loudness of the heart sounds, many of these problems will be dealt with satisfactorily.

In what has just been said as to the important part which interference with pulmonary circulation plays in causing the type of dilatation of the right ventricle, which is now under discussion, it is not intended to deny the part which systemic overstrain may play in these cases.

For there is no doubt that the severity of the cardiac failure in many of the cases of anæmia, with which we are dealing, was due to the fact that in spite of weakness and malnutrition, the girl had to continue to work for her living and often at work requiring considerable exertion. Where the heart was weak, owing to the anæmia, the performance of ordinary work would result in overstrain for the cardiac muscle, and the result would be much the same as that produced by excessive work upon the normal heart muscle.

This fact will be made clear by comparing the type of dilatation in simple overstrain, apart from anæmia, which is described in Essay VI, p. 308, with the type of dilatation with which we are now dealing.

Having now discussed from a clinical standpoint the nature of the dilatation of the right ventricle which occurs in anæmia, and its probable cause, the subject must next be considered from a pathological standpoint, not only as regards the alteration in the shape of the ventricle itself and its effect upon adjacent structures, but also as regards the changes in the circulation which are associated therewith. The chief of these changes is that whereby the pulmonary artery systolic murmur is produced.

The theory of production of this murmur is dealt with from the pathological side in the following essay, which was read before the Pathological Society of London in the year 1899, and is here given complete as it appears in their *Transactions*.

PATHOLOGICAL ASPECTS OF UPWARD DILATATION OF THE RIGHT VENTRICLE[1]

The aim of this paper is twofold: firstly to demonstrate the change in shape which the upper part of the right ventricle undergoes when it dilates; and secondly, to show the changes in the pulmonary artery which result from this upward dilatation of the right ventricle, and point out the bearings which they have upon the causation of the systolic murmur which is heard over the pulmonary artery in cases of cardiac overstrain and anæmic dilatation of the heart.

---

[1] Read before the Pathological Society of London, April 18, 1899, and reprinted from the *Transactions of the Pathological Society of London*, 1899.

PHOTOGRAPHS OF CARDIAC VENTRICLES FROM ABOVE AFTER REMOVAL OF THE AORTA, PULMONARY ARTERY, AND THE TWO AURICLES

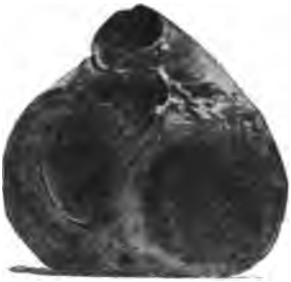


FIG. 59—NORMAL HEART.

It will be noticed that little of the anterior wall can be seen, and that the base of the pulmonary artery is in the photograph almost vertically over the aortic valves.

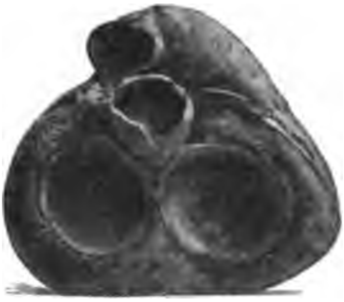


FIG. 60.—HEART WITH SLIGHTLY DILATED RIGHT VENTRICLE.

It shows that the anterior wall of the right ventricle bulges forward in front of the aorta, and pushes the origin of the pulmonary artery to the left of its normal position.

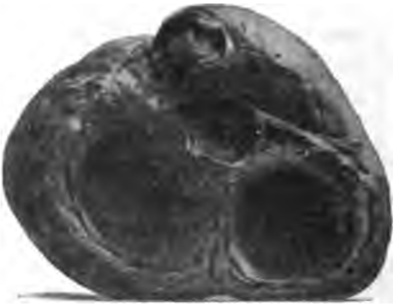


FIG. 61.—HEART FROM AORTIC AND MITRAL DISEASE, WITH DILATATION AND HYPERTROPHY OF BOTH LEFT AND RIGHT VENTRICLES.

It shows very marked forward bulging of the anterior wall of the right ventricle and the considerable displacement of the pulmonary artery to the left. It also shows that the pulmonary valves and upper part of the right ventricle are not in the plane of the auriculo-ventricular septum as in Figs. 59 and 60, but lie in a higher plane —i.e. are in the photograph projecting forwards towards the observer.

Figs. 62 and 63.—Lateral View of Distended Hearts. (Drawn from photographs, three-sevenths natural size.)

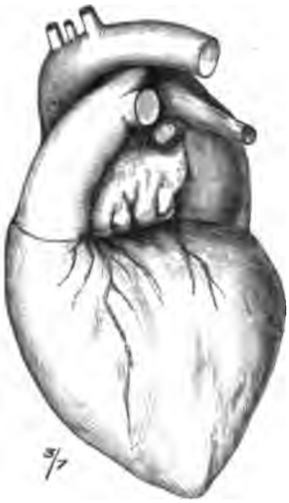


Fig. 62.—Normal Heart.

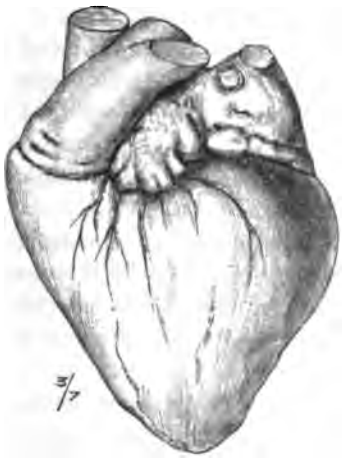


Fig. 63.—Heart with Slight Dilatation of the Right Ventricle Upwards.

It shows the elevation of the pulmonary valves and consequent shortening of the artery, and also the altered direction of the artery relatively to the main axis of the right ventricle.

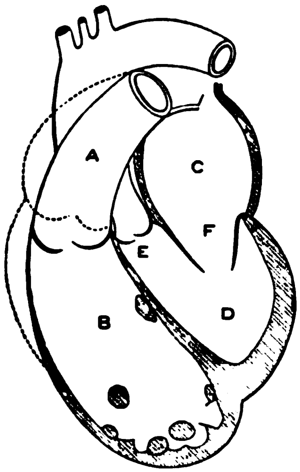


Fig. 64.—Semi-diagrammatic View of a Normal Heart as seen in Section through the Pulmonary Artery and Right Ventricle.

To illustrate the shortening and dilatation of the pulmonary artery which results from upward dilatation of the right ventricle. The dotted line represents the change that takes place.

This paper is illustrated by the study of three hearts :—

1. From a man who died as the result of an accident. This heart may be considered normal.

2. From a woman, aged forty-eight, who died suddenly from hemiplegia with cardiac failure, and who was found to have a congenital defect in the interauricular septum. In this case the right ventricle and right auricle are rather dilated, and the left are approximately normal.

3. From a girl aged fifteen, who died from cardiac failure, the result of disease of both aortic and mitral valves. In this case the right ventricle is greatly dilated as well as the left ventricle and auricle.

The hearts were distended with hard paraffin prior to their removal from the body, with the exception of that from Case II, fig. 60, which was not distended *in situ.*

As to the pressure under which the injection was made, I employed an ordinary injection syringe and did not use a manometer or other pressure gauge, but was careful not to overdistend the hearts. I cannot, therefore, state the pressure employed, nor do I consider it a point of importance in the present connection, since my object is to illustrate one of the ways in which the ventricle dilates, and not the degree of dilatation that occurs. I, nevertheless, am confident that the specimens which I am showing are truthful in this respect also.

Figs. 59 to 61 show the ventricles isolated by the removal of the auricles and aorta and pulmonary artery.

Comparison of the two dilated right ventricles thus isolated, with that of the normal heart shows very clearly the change in shape which takes place when the right ventricle dilates upwards in the manner so well described by Dr. Foxwell in his 'Essays on Heart and Lung Disease.'

In a view of the isolated ventricles from above, as in the photographs (figs. 59, 60, and 61), the dilated upper portion of the right ventricle is seen as a distinct prominence bulging forwards beyond the normal limits. Not only does it project anteriorly, as is well shown in the photographs of hearts, figs. 60 and 61, but it also projects upwards beyond the plane of the auriculo-ventricular septum, and so comes to overhang the commencement of the aorta. This was well seen in the case of the heart shown in fig. 61, when the isolated ventricle was viewed from the side. It can be seen in fig. 61.

I do not show these specimens of upward dilatation of the right ventricle as an unusual condition, but as good demonstration of a very common condition; for although this type of dilatation seems to be but seldom referred to in current literature, I believe that if it be carefully looked for clinically and in the post-mortem room it will be found to be extremely frequent in cases of cardiac failure from a great variety of causes.

It is, I believe, the ordinary type of dilatation in youth and early adult life, for at this period dilatation of the right ventricle upwards and to the left usually precedes and exceeds in amount any dilatation of the ventricle to the right.

In adults this upward dilatation seems to be, as a rule, *less* marked than dilatation to the right of the sternum, and after the age of forty-five or so it seems to be decidedly rare; for in the later years of life, the dilatation is towards the right side, and the conus arteriosus yields but little.

The upward dilatation is often very extreme in youth and adolescence. For example, the relative dullness of the heart, shown in fig. 61, commenced above the second rib on the left side and extended to the nipple line in the third left interspace, and after death, some months subsequently, the pulmonary valves were found to lie at the level of the first rib. Such a degree of dilatation is not very uncommon.

I now come to the consideration of the effect which this upward extension of the conus arteriosus has upon the pulmonary artery. Its effect is twofold: firstly, the artery is shortened; secondly, the direction of its course is altered.

Firstly, as to the shortening of the pulmonary artery. It is evident that the increase of the upper part of the right ventricle upwards and to the left must carry the origin of the pulmonary artery upwards with it; and since the course of the artery is normally upwards from the pulmonary valves, any elevation of the point of origin must shorten the course of the artery. This is shown by comparing fig. 62 with fig. 63, and also by the diagram.

When, as is often the case, the pulmonary valves lie under the second rib instead of under the third, this shortening must amount to ¾ inch or more. Taking the normal length of the artery at 2½ inches, this would mean a shortening of 30 per cent. The effect of this shortening must be to relax the walls of the artery.

This relaxation may be seen post-mortem as a distinct wrinkling of the arterial wall in extreme cases (see fig. 63). More often, however, the elastic contractility of the vessel prevents any distinct wrinkling. Although the relaxation of the vessel wall may not show after death when the artery is empty, it cannot fail to cause some distortion during life. The relaxed wall is sure to yield abnormally to the blood pressure, and dilatation of the artery will result. There will, therefore, be both shortening and dilatation of the pulmonary artery, and the two combined will tend to make the artery become more spherical than normal. In other words, when the shortening is considerable, there will be an aneurysmal bulging of the pulmonary artery under the influence of the blood pressure.

And now *as to the altered direction of the pulmonary artery.* Taking the normal course of the first part of the artery as being obliquely upwards and backwards, it is evident that if its point of origin is raised its course will become more horizontal than normal. When, as often happens, the pulmonary valves lie under the second rib instead of under the third, the alteration in the course of the artery is considerable.

This altered course means an alteration in the axis of the pulmonary artery as compared with the axis of the ventricle, and instead of the artery being fairly in line with that axis, as it ought to be, it will be at an angle to it. I have frequently noted this change in the direction of the pulmonary artery, post-mortem, more especially if the ventricle happened to be distended. Under such circumstances the sudden dipping backwards of the artery is most noticeable. Thus, in the case of the heart shown in fig. 61, I noted at the post-mortem that the first part of the pulmonary artery was at right angles to the longitudinal axis of the right ventricle, instead of being nearly in the same line as in a normal heart.

In fig. 64, a semi-diagrammatic view is given of a section through the centre of the pulmonary artery and right ventricle to illustrate the distortion of the former. It is not easy to represent the change in the direction of the artery of which we are now speaking in this diagram, because it is accompanied by a movement of the pulmonary artery and valves to the left of the normal position—i.e. out of the plane of the diagram.[1]

This lateral movement of the artery is well shown in the photographs on p. 248. In the normal heart (fig. 59), the pulmonary valves are seen to be almost vertically over the aortic valves. In both of the other hearts the pulmonary valves and base of the pulmonary artery are seen to lie considerably to the left of their normal position.

The practical result of this alteration in the direction of the pulmonary artery as compared with the axis of the ventricle, is as follows: When the ventricle contracts, the blood is thrown against the anterior wall of the pulmonary artery, instead of being thrown in the direction of its lumen. This will be seen on comparing fig. 62 with fig. 63.

Here, then, we have two conditions eminently favourable to the production of an aneurysmal murmur. We have a pulmonary artery that becomes more globular than normal under the influence of the blood pressure, and we have the blood thrown into it so as to strike one of its walls—conditions likely to bring about a murmur-producing eddy in the dilated artery.

These two factors, the increased distensibility of the pulmonary artery, and the alteration of its axis, as compared with that of the right ventricle, are, I believe, of prime importance in the causation of the common systolic murmur which is audible over the second left interspace in cases of cardiac overstrain and cardiac debility in anæmia and other diseases: in other words, I feel satisfied, from clinical and pathological evidence—to which I cannot refer within the limits of this paper—that the type of dilatation of the right ventricle with which this paper deals is the pathological condition which produces the common pulmonary systolic murmur of anæmia and overstrain, and that the mechanism is such as I have suggested.

---

[1] The attachment of the posterior part of the pulmonary artery and of the adjacent part of the right ventricle to the anterior part of the aorta and left ventricle is not sufficiently firm to prevent such a shifting of the pulmonary artery from taking place.

I do not say, however, that upward dilatation is sufficient by itself to produce this murmur in all states of the circulatoin, for I know that such is not the case. Extreme degrees of upward dilatation of the right ventricle can exist without producing any basal murmur (*vide* p. 314 and fig. 118).

Upward dilatation of the ventricle does, however, give rise to the possibility of an aneurysmal murmur being produced in the pulmonary artery.

When the conditions of the circulation are favourable, an aneurysmal murmur is produced in the artery, and even an aneurysmal thrill. When they are not favourable no murmur will be produced.

This, then, is the bearing which I believe upward dilatation of the right ventricle has upon the production of the pulmonary systolic murmur of anæmia and cardiac debility.

Having discussed the pathological changes which are noticeable in anæmic heart failure, and the theory which they suggest as to the cause of the hæmic murmur which is heard in these cases, the clinical character of this murmur must now be discussed in some detail in order fully to test the applicability of the theory which has been propounded as to its origin in a dilated pulmonary artery.

PULMONARY ARTERY SYSTOLIC MURMUR

In the first place, this murmur—which was formerly called the basal hæmic murmur of anæmia—must be defined. It is a systolic murmur heard at the base of the heart in cases of anæmia with heart failure, and its point of maximal loudness is usually in the second interspace to the left of the sternum and over the situation of the origin of the pulmonary artery.

ITS FREQUENCY.—In anæmia this pulmonary murmur is heard in the majority of cases which show signs of cardiac failure. The actual figures obtained from the series of cases here dealt with are as follows :—

Out of 350 consecutive cases of anæmia, in 63 no notes were taken, and in 45 the heart was normal, leaving 242 cases of heart failure of which notes were taken.

Out of these 242 cases 8 are not available, as no note was made as to the presence or absence of a pulmonary murmur. Out of the remaining 234 cases a pulmonary murmur was present in 227 instances, or exactly 97 per cent. of the cases.

It can therefore be asserted that a pulmonary artery systolic murmur is *usually* present in cases of the heart failure which accompanies the simple anæmia (and chlorosis) of adolescence.

The clinical features of this murmur will now be discussed under the headings :—

Its character : 'What' is it ?

Its time relationships : 'When' is it heard ?

Its point of maximal loudness and area of audition : 'Where' is it heard ?

The factors at work in its causation : 'How' is it produced ?

THE CHARACTER of the pulmonary systolic murmur.—This murmur is usually somewhat low in tone, and resembles a faint aneurysmal murmur more closely than it does the higher pitched and more blowing murmur of mitral regurgitation. As regards its loudness it shows a wide range, for not only does its loudness in any particular case vary very readily with changes in the heart's activity, and even with the attitude the patient is in during auscultation, but it also shows a wide range of loudness according to the severity of the anæmia present. In a slight case the murmur may be soft, low-toned, and barely audible; and in a more severe case it may be so loud as to be heard over the greater part of the chest and even be accompanied by a well-marked vibratile thrill such as that felt over an aneurysm.

Moreover, a murmur of this degree of intensity may be found to disappear entirely in a few weeks, coincidently with the general improvement of the patient.

This fact of the ready variability of the murmur and that it does not occur in any particular case with the comparatively regular loudness which we associate with such systolic murmurs as those produced at the mitral or aortic orifices is of very considerable importance.

VARIABILITY of the pulmonary systolic murmur with CHANGE OF ATTITUDE of the patient.—This is one of the most marked features of this murmur.

Often when the murmur is faint or almost inaudible when the patient is standing it becomes quite distinct or even loud on lying down (see figs. 65 and 66). It is the rule that this murmur is distinctly louder when the patient is lying down than it is when sitting up or standing. Statistics on this point are as follows: In fifty-two cases where this murmur was audible, the effect of change of the patient's position was noted, with the result that in forty-seven instances the murmur was distinctly louder when the patient was recumbent. In three cases there was no definite change and in two cases the murmur was distinctly louder when the patient was in the erect attitude.

CASE SHOWING LOUD PULMONARY MURMUR WHEN RECUMBENT AND NO MURMUR WHEN ERECT

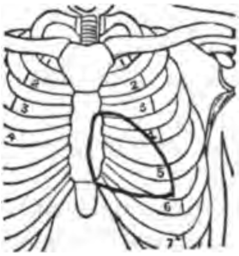


FIG. 65.

Eliza C., aged twenty-three, March 31, 1891. Showing normal heart in erect attitude. No dilatation and no murmur.

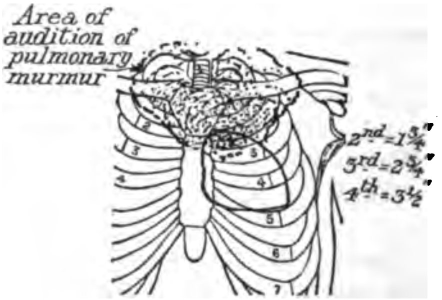


FIG. 66.

Eliza C., March 31, 1891. Showing upward displacement and dilatation of the heart on lying down, with a loud pulmonary murmur audible into vessels of the neck, also 1¾ inches of dullness in second left space and a systolic murmur at the apex.

These two cases were, however, exceptional in other ways, and will be dealt with later on (*vide* p. 300).

Thus in 96 per cent. of the cases examined in this series the murmur was increased in loudness in the recumbent attitude, and this statement quite coincides with what is usually found when this murmur is listened for in these two different attitudes. This question is again referred to below (*vide* p. 266).

VARIABILITY with slight changes in the heart's activity.—Further, the increase in the loudness of the murmur when the heart is acting excitedly is somewhat in excess of that usually observed in the case of other cardiac murmurs, under similar circumstances. Nervous excitement, too, will increase this murmur more than it will increase organic cardiac murmurs. These facts must be borne in mind when testing the effect of change of attitude; for the exertion of sitting up or walking to a couch and lying down will in some cases decidedly increase the loudness of the murmur for some few seconds (for illustration, *vide* p. 268).

Again, if the loudness of the murmur be carefully noted, and especially if some means be used for testing its power of penetration through some resisting medium (*vide* p. 413), it will often be found that there is a slight respiratory variation in its loudness, and that the murmur with the cardiac beat occurring at, or just after, the end of inspiration is a little louder than the murmur occurring with the beats during the remainder of the respiratory cycle.

TIME AND MODE OF OCCURRENCE of the pulmonary systolic murmur.—The murmur, as its name implies, is systolic in rhythm, accompanying but not replacing the first sound of the heart, although, when at all loud, it may seem to do so.

When the murmur appears to replace the first sound at the base of the heart, it will be found, nevertheless, on listening lower down, over the fourth and fifth interspaces, that as the murmur lessens in loudness the first sound becomes more distinct, until a point may usually be found, towards the lower end of the sternum, where the right ventricular first sound may be heard pure and free from murmur.

If it were possible to record the exact time of occurrence of the murmur, it would, I feel sure, be found to begin a fraction of a second later than the commencement of a systolic regurgitant murmur.

The characteristic of the murmur to be next discussed is the place or area *where* it is heard.

POINT OF MAXIMAL INTENSITY, AREA OF AUDITION AND DIRECTION OF CONDUCTION of the pulmonary systolic murmur. —The point of maximal intensity of the murmur is usually found to be at the sternal end of the second left interspace. Its exact position varies somewhat in different cases, and this variation is apparently due to the fact that the murmur is best heard over the situation of the pulmonary valves and the adjacent part of the pulmonary artery, and the position of these structures is not constant, but is found to vary according to the amount of dilatation of the right ventricle which is present in any particular case. In some cases the pulmonary valves lie close to their normal situation under the sternal end of the third costal cartilage. Whereas, when there is much upward dilatation of the ventricle, they may lie in the second left interspace as much as one inch or more away from the sternum. Also they may lie at a much higher level than normal and be under the second rib or even higher. Under such conditions the point of maximal intensity of the murmur (or the 'P.M.I.,' as it may be called) will be correspondingly farther from the sternum or higher than normal. Practically, it will be found that the P.M.I. of the murmur is, as a rule, the same as the P.M.I. of the pulmonary second sound.

As regards the situation of the P.M.I. of the murmur, it can sometimes be noted that it changes a little with the change in the amount of the cardiac dullness in the second interspace, which takes place when the patient lies down.

It may not infrequently be noticed that the P.M.I. of the pulmonary second sound is definitely at a higher level in the recumbent than in the erect attitude, and occasionally this same observation may be made with the less easily definable point of maximal intensity of the pulmonary systolic murmur.

THE AREA OF AUDITION of the murmur.—The size of the area over which the murmur can be heard depends, naturally, upon the loudness of the murmur.

When faint, its area of audition is only that of a half-crown or five-shilling piece, and is situated just over the pulmonary valves; when louder, the area extends upwards and to the left towards the centre of the clavicle, and is elliptical in shape (*vide* fig. 71, p. 620).

A point of importance is that the murmur is not well heard below the fourth rib unless it be very loud. If, when the murmur is not well conducted elsewhere, it appears to be heard just to the left of the sternum in the third, fourth, and fifth interspaces, it may be taken for granted that what appears to be a propagation downwards of the pulmonary systolic murmur is really due to the presence of a tricuspid regurgitant murmur, and is not a sign that the pulmonary artery murmur is audible over these interspaces.

When the pulmonary murmur is loud it may have a fairly wide area of audition downwards as well as on both sides of the sternum, and an exceptionally loud murmur may be audible over the whole of the upper part of the chest and the cardiac region. There is one point of diagnostic importance to be noticed with regard to the conduction of this murmur and that is that when the murmur is loud it may be conducted into the carotid and subclavian arteries and so cause doubt as to whether a true aortic systolic murmur be present or not.

When a thrill is present it, too, may be just palpable in the carotid arteries. The reason for this direction of conduction is to be found in the anatomical relationship of the pulmonary artery and the aorta. If this be studied it will be seen that if the pulmonary artery were to be distended and altered in position so as to lie somewhat higher than normal it would come into close relationship with the inner side of the upper part of the ascending arch of the aorta and of the commencement of the transverse arch. It can therefore easily be understood that when the blood in the distended pulmonary artery is vibrating sufficiently to cause a loud murmur, or even the palpable thrill that can, in exceptional cases, be felt, these vibrations must involve that part of the aorta against which the pulmonary artery is pressed and by throwing the blood in the aorta into vibration lead to the

PULMONARY MURMUR AND UPWARD DILATATION OF RIGHT VENTRICLE, BOTH MODERATE. (AREA OF AUDITION OF THE MURMUR SHOWN BY DOTTED SHADING.)

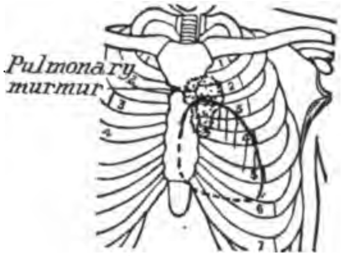


FIG. 67.

Nellie H., aged nineteen.

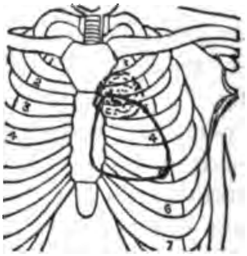


FIG. 68.

Sarah J., aged twenty.
Faint murmur.

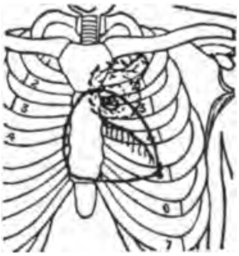


FIG. 69.

Jane T., aged seventeen.
Murmur very faint.

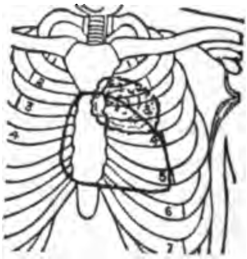


FIG. 70.

Rose K., aged fifteen.

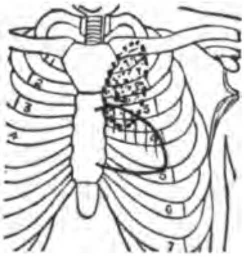


FIG. 71.

Minnie P., aged sixteen. Murmur moderately loud. Right ventricle third sound present. When erect, first sound louder; pulmonary murmur fainter.

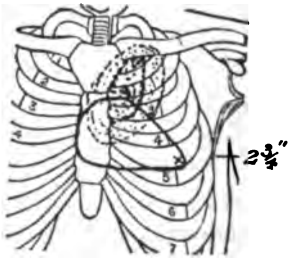


FIG. 72.

Annie D., aged sixteen. Pulmonary murmur moderately loud. Faint mid-diastolic mitral present.

LOUD PULMONARY MURMUR WITH CONSIDERABLE DILATATION, AND FIGS. 73, 74, AND 76 WITH FUNCTIONAL MITRAL AND TRICUSPID MURMURS AS WELL

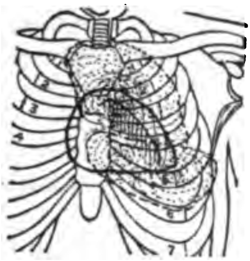


FIG. 73.

Gertrude L., aged nineteen. Loud pulmonary also mitral and tricuspid murmurs. When erect, no tricuspid and a fainter pulmonary murmur.

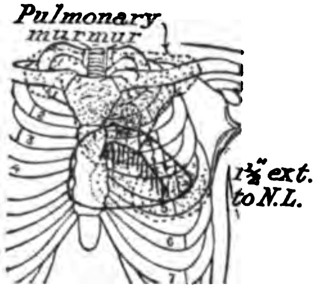


FIG. 74.

Jane B., aged twenty. Loud pulmonary and mitral murmurs and faint tricuspid.

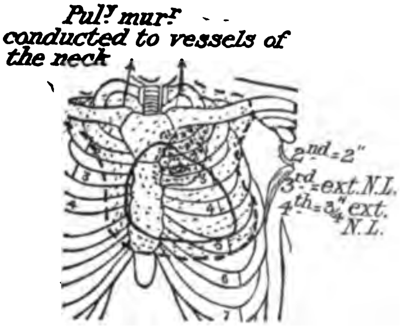


FIG. 75.

Ellen H., aged twenty. Considerable dilatation and loud and widely conducted pulmonary murmur. After a few weeks' treatment the heart was normal again.

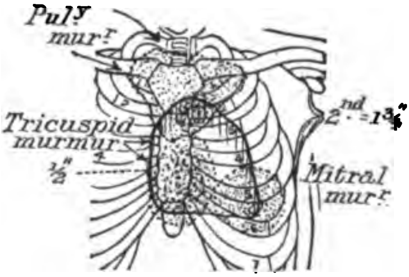


FIG. 76.

Eliza B., aged nineteen. Great dilatation; loud pulmonary, sharp blowing tricuspid, and soft mitral murmurs.

conduction of the murmur along the arteries. Careful examination will confirm this theory as to the arterial conduction of the pulmonary systolic murmur, for it will be noticed that the murmur is rather better heard in the left carotid and subclavian arteries than it is in the right. This appears to be due to the fact that the vibrations coming from the pulmonary artery are not so likely to involve the blood passing up the innominate artery as they are the blood which remains a little longer in the arch of the aorta and comes more in contact with that part of the vessel against which the vibrating pulmonary artery is pressed.

Figs. 67 to 76, taken from my out-patient case-books, show the variations in the area of audition and direction of conduction of this murmur in different cases.

Having discussed the chief characteristics of this murmur, the theoretical consideration as to its mode of production must now be taken up.

Before doing so, however, I must say a few words as to the important work which the late Dr. Arthur Foxwell did upon this subject. Those who compare what I have here written upon the heart in anæmia with his published writings will see that I am travelling upon much the same lines that he did. It was his work which stimulated me twenty-five years ago to devote much of my time to this subject, and much of what I am now writing consists in filling in the main outlines of the subject which were sketched out by him.

As regards the cause of the pulmonary systolic murmur, I am partly in agreement with him and partly not.

As regards the part played by relaxation of the wall of the pulmonary, I can fully confirm all he said, but I do not consider that the other factor he spoke of is so important as he seemed to think: I refer to the alteration in level of the valve-plane in the pulmonary artery. In the undistended heart post-mortem, it is certainly noticeable that the anterior portion of the pulmonary valve lies at a higher level than its posterior part. And I have seen a case where the top of the anterior cusp of the valve was under the upper border of the first rib, whereas the top of the posterior cusp was under the lower border of the same rib. This would seem to confirm Dr. Foxwell's view. When, however, such a heart as this is distended, it is found that this alteration in the plane of the valve is simply due to the altered direction of the artery, and as the valve is still at right angles to the direction of the blood-flow there is nothing in this alteration likely to cause a murmur.

In this connection it is interesting to note that the union between the posterior wall of the pulmonary artery and the anterior wall of the aorta is not very firm in these cases, and that the relative position of the two orifices is capable of a certain amount of variation (*vide* p. 253, and figs. 60, 61).

Mode of Production of the pulmonary systolic murmur.—The various clinical characteristics of this murmur must now be reviewed in connection with the theory as to its production already advanced—namely, that it is a murmur produced during systole in an aneurysm-like over-distension of the pulmonary artery which is rendered possible by the relaxation of its walls, brought about by the upward dilatation of the conus arteriosus of the right ventricle.

1. The murmur is evidently a right ventricle murmur and not a left ventricle one. This is shown—

(1) By its ready variability—

(*a*) With change in the attitude of the patient as already pointed out (p. 256);

(*b*) With slight changes in the strength of the heart's action—e.g. on excitement and slight exertion and with respiration; and—

(2) Its right ventricular origin is also suggested by its lowness of tone.

2. The murmur is not produced at the tricuspid orifice because of its relationship to the right ventricular first sound; for when the murmur is quite well marked in the second interspace the tricuspid first sound can as a rule be heard to be quite pure and free from murmur on listening over the sternal ends of the fifth and sixth interspaces.

3. The ready variability of the murmur shows that it is not due to any permanent damage, but suggests that it is due to some transient cause—such as the over-distension of some elastic structure—that can readily return to its normal size after the disturbing factor is withdrawn: such, indeed, as the over-distension of the elastic pulmonary artery and the readily dilatable part of the right ventricle.

4. Its point of maximal intensity, area of audition, and direction of conduction are all in favour of its pulmonary artery origin.

5. Its causation must be closely connected with the upward dilation of the right ventricle, because within certain

well-defined (and easily explainable) limits the loudness of the murmur is proportional to the amount of upward increase of the cardiac dullness which is present.

This is shown by the following table giving the amount of dilatation present, together with the loudness of the murmur, in a consecutive series of over 200 cases of anæmic heart failure upon which 274 observations were made.

Increased Loudness of the murmur accompanied **Increased Upward Dilatation** of the heart.—The relationship which the loudness of the pulmonary artery systolic murmur bears to the amount of upward dilatation of the right ventricle is well shown in the following table, which gives an analysis of 274 observations upon 204 cases of anæmia which showed upward dilataion of the heart, and a pulmonary systolic murmur either separately or, more usually, combined.

In this table very loud murmurs are those heard widely over the chest and well conducted into the vessels of the neck.

LOUDNESS OF THE PULMONARY ARTERY SYSTOLIC MURMUR IN RELATION TO THE UPWARD DILATATION OF THE RIGHT VENTRICLE

Amount of dilatation of the right ventricle upwards and to the left	No murmur	Very faint murmur	Faint	Moderately loud murmur	Loud	Very loud	Total observations	Total cases
'Slight'	1	1	1	6	—	—	9	8
'Present'	2	—	2	3	4	—	11	11
'Considerable' . . .	—	—	—	—	—	1	1	1
¼ inch to ½ inch in second space	2	3	6	7	5	—	23	17
¾ inch in second space . .	3	6	5	2	8	2	26	20
1 inch ,, ,, . .	4	3	8	17	16	3	51	32
1¼ inch ,, ,, . .	1	0	6	8	8	4	27	21
1½ inch ,, ,, . .	—	—	3	11	8	7	29	23
1¾ inch ,, ,, . .	—	—	1	1	2	4	8	7
2 inches ,, ,, . .	—	—	2	7	6	4	19	14
More than 2 inches in second space	—	—	—	1	1	1	3	3
3 inches or more ,, ,, ,,	—	—	1	1	—	—	2	2
None in second but 2 in. in third	1	1	3	7	2	—	14	10
,, ,, ,, 2½ ,, ,,	1	1	2	3	—	2	9	7
,, ,, ,, 3 ,, ,,	—	—	—	—	—	1	1	1
None in second but some in third	1	—	2	1	—	—	4	3
No dilatation	10	4	10	5	3	0	32	19
No notes as to dilatation . .	1	—	1	3	—	—	5	5
Total	27	19	53	83	63	29	274	204

Loud murmurs are those conducted up to the clavicle and outwards along the first part of the subclavians. Sometimes, however, a loud murmur may be fairly localised. Moderately loud, faint, and very faint, murmurs are those having smaller areas of audition and the less degrees of loudness which their description implies.

In this table, if attention be confined to those cases which show less than two inches of relative cardiac dullness in the second left interspace, it is evident that the loudness of the murmur increases with the increase in the amount of dullness. When, however, there is a larger amount of dullness than this, the same continuous increase in the average loudness of the murmur is not noticeable, and occasional cases are met with where the murmur is faint or even absent.

These cases will be explained later, and in the meantime it can be stated, having regard to the above statistics, that for moderate amounts of dilatation the loudness of the pulmonary systolic murmur is proportional to the amount of upward dilatation of the right ventricle which is noticeable in the second left interspace.

This same law is also confirmed by the few cases tabulated where there was no increase of dullness in the second interspace, but where there was a broadening of the cardiac dullness in the third interspace. The following summary of the above table shows these facts rather more clearly :—

Loudness of Murmur

Amount of dullness	Observations	Faint and very faint	Moderate	Loud and very loud	Loud	Very loud
Under 1 inch in second space	49	22 = 40 %	9 = 18 %	15 = 30 %	26 %	4 %
1 inch of dullness in second space	51	11 = 22 %	17 = 34 %	19 = 38 %	32 %	6 %
1¼ inch to 1¾ inch in second space	64	10 = 15 %	20 = 31 %	33 = 51 %	28 %	15 %
2 inches and more in second space	24	3 = 12½%	9 = 37 %	12 = 50 %	29 %	20 %

The next point in confirmation of the theory here advanced as to the causation of the pulmonary artery murmur is that whenever the amount of cardiac dullness in the second or third interspace shows a *temporary* increase from change in the attitude of the patient or from excited action of the heart, the murmur also is correspondingly increased in loudness.

The increase in the cardiac dullness which results from change of attitude in these cases of upward dilatation of the ventricle is usually very marked. This increase of the cardiac dullness is probably due mainly to the action of gravitation acting in two ways: firstly, by causing the heart to assume a somewhat lower position in the erect than in the recumbent attitude, as is shown by the alteration in the level of the apex (*vide* p. 312); secondly, the heart being largely fixed by the attachment of its pulmonary artery and aorta to the roots of the lungs (and to the tissues of the posterior mediastinum), the weight of the heart increases to a certain extent the tension of the anterior wall of the pulmonary artery and the adjacent part of the anterior wall of the ventricle when the patient is standing, and this tends to restrain dilatation. When lying, this tension is removed, and slightly more dilatation takes place therefore in that position.

The following are some notes of the amount of this increase of the cardiac dullness in the second left interspace when in the recumbent as compared with the erect attitude.

In fourteen cases where there was no dullness when the patient was standing, the following amounts of relative dullness appeared in the second interspace when the patient lay down: ¼ inch in one case; ¾ inch in four cases; 1 inch in five cases; 1¼ inches in three cases, and 1½ inches in one case. In ten other cases, where there was some dilatation when standing, there was an increase of from ¼ to 1½ inches. In two of these there was 1 inch—namely, from ¼ inch to 1¼ inches, and from 1 inch to 2 inches; and in two there was increase of 1½ inches—namely, from 1 inch to 2½ inches. The twenty-four cases may be summed up as follows:—

Two cases showed an increase of ¼ inch on lying down.
One case ,, ,, ½ ,, ,,
Eight cases ,, ,, ¾ ,, ,,
Seven ,, ,, ,, 1 ,, ,,
Three ,, ,, ,, 1¼ ,, ,,
Three ,, ,, ,, 1½ ,, ,,

The fact that most of these cases show large amounts of increase of dullness is due to the fact that the cases seen were hospital out-patients, and the greater degrees of increase were more apt to be noted than the smaller amounts which required more careful observation for their detection.

In addition to these twenty-four, in ten other cases the *fact* of an increase in the amount of relative dullness in the second left interspace was noted, but its amount was not stated.

These cases are simply illustrative of the increase of dullness; for if it had been more systematically noted it would have been recorded as occurring in most of the patients where upward dilatation of the right ventricle was well marked.

A similar increase of the cardiac dullness is also often noticeable when the heart begins to act rather more excitably from exertion or from nervousness. With the increase of dullness produced in either of these ways, there is almost invariably an increase in the loudness of the pulmonary systolic murmur.

The following facts illustrate this point:—

The effect of change of position upon both the amount of relative dullness in the second left interspace and the pulmonary systolic murmur was noted in twenty-seven instances, and in twenty-five of these both the murmur and the dullness were increased. Of the remaining two there was no distinct change in one, and in the other the murmur was louder in the erect attitude. This case is discussed later (*vide* p. 303).

This increase of the loudness of the murmur in the recumbent attitude is so characteristic as to be almost diagnostic. The effect of attitude upon the murmur was also definitely noted in twenty-eight other cases, although no note was made as to the presence of any accompanying increase in the cardiac dullness. Out of these twenty-eight cases the murmur was definitely increased on lying down in twenty-four cases. In two of these there was no change, and in two the murmur was slightly louder when the patient was standing up.

Thus, out of the fifty-five cases noted, the murmur was louder in the recumbent attitude in forty-nine, and of the remaining six there was no change in its loudness in three;

and in the other three it was louder in the erect than in the recumbent attitude.

Increased Loudness Due to Excited Action of heart. —Increased loudness of the pulmonary artery murmur is also commonly noticeable under conditions somewhat allied to those just mentioned—namely, during the slight increases in the heart's activity which result from exertion or from nervous excitement. Such increase in the heart's activity will tend to increase the loudness of the murmurs in two ways —namely, by increasing the relaxation of the artery (owing to the increased upward dilatation of the ventricle) and also by temporarily increasing the blood pressure in the pulmonary artery whereby the over-distension is caused.

Cases showing an Inconstant and Variable Pulmonary Murmur

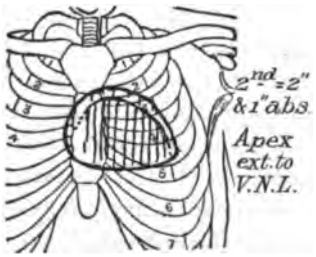


Fig. 77.

Maria G., aged twenty-one. Showing size of heart during full expiration. Pulmonary murmur sometimes very loud, sometimes almost absent.

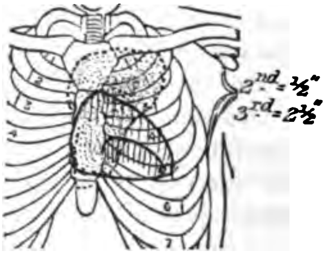


Fig. 78.

Eliza S., aged twenty. Showing amount of dilatation and area of murmur on lying down immediately after exertion. None when erect.

Here are two such cases taken from the notes of my out-patient cases.

A girl of eighteen who had paid two fortnightly visits to the hospital and was rapidly improving, but still had one inch of relative dullness in the second left interspace, had a loud pulmonary murmur when she first lay down on the couch for examination, but it rapidly got less and, in the course of two or three minutes, almost entirely disappeared. Also in a young woman aged twenty-one, who had a very considerable amount of right ventricular dilatation (*vide* fig. 77: the murmur is noted as 'being very loud at times [during the ten or fifteen minutes that the examination of the heart lasted] and at times almost inaudible.' Slight variations in the excitability of the heart's action being sufficient to make this great difference in the loudness of the murmur (see also fig. 78).

Other clinical features which tend to support the view here advocated as to the pulmonary murmur being due to an abnormal distension of the pulmonary artery are the frequent association of a loud pulmonary second sound with this murmur and also the fact that these cases do not as a rule show any signs of tricuspid regurgitation, for leakage through the tricuspid valve would, as has already been pointed out, tend to prevent the intraventricular pressure from rising to a sufficient height to cause upward dilatation of the ventricle and over-distension of the pulmonary artery in these cases.

Where this type of dilatation of the right ventricle in adolescents is secondary to severe valvular disease of the left side of the heart, it is, however, common to find tricuspid regurgitation and dilatation of the ventricle to the right, associated with upward dilatation of the ventricle and a pulmonary artery murmur.

Having now referred to the clinical facts which support the main thesis here advanced that the basal hæmic murmur of anæmia is due to the over-distension of the pulmonary artery which is brought about by the upward dilatation of the right ventricle, we must now go rather more fully into the various factors concerned in order to prepare the way for the explanation of the exceptions to the rule laid down which are from time to time met with.

In noting the presence or absence of a pulmonary artery murmur in a long series of cases showing dilatation of the right ventricle upwards, the observer cannot fail to be struck with the seeming want of certainty as to whether any particular type of case will have a loud pulmonary murmur or not.

For instance, I have stated that dilatation of the right ventricle upwards so as to cause increased dullness in the second left interspace is the usual cause of the murmur, and yet in some of the cases where the dilatation is extreme no murmur is audible. Again, whereas in one case where this upward dilatation is moderate in amount the murmur is very loud; in another apparently similar case it is faint or absent (see figs. 79 to 81). And again, a patient who is very ill and breathless may have a faint murmur, and yet at a subsequent visit, when undoubtedly better, there may be a loud murmur with or without an increase instead of the lessening in the amount of dilatation which her improvement would suggest.

Moderate Upward Dilatation of Right Ventricle, but no Pulmonary Murmur. (The Cause of the Absence of a Murmur was not Investigated.)

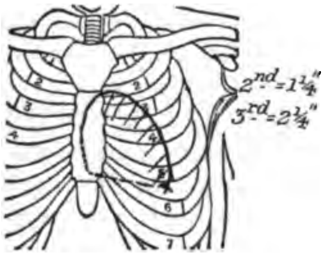


Fig. 79.

Kate L., aged twenty-eight. Moderate dilatation, but no pulmonary murmur.

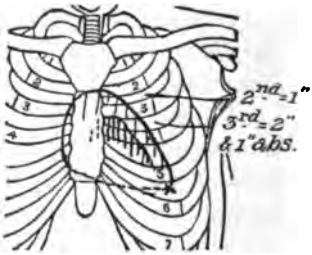


Fig. 80.

Sarah S., aged eighteen. Dyspnœa, with pulsation and some dullness in second space, but no murmur.

In some cases the murmur is present where there is no dilatation.

Again, although the murmur is as a rule, greatly increased when the patient lies down, an occasional case is met with where this is not the case and the murmur may even be louder when the patient is standing up.

These exceptions to the rule do not, however, shake the evidence which has been adduced as to the cause of this murmur, and they can quite easily be shown to be confirmatory of it.

Factors Concerned in the Production of the Pulmonary Artery Systolic Murmur

Several factors must be recognised as being concerned in the production of the pulmonary systolic murmur, and unless their relative importance is recognised it will not be possible to understand the seeming uncertainty as to whether this murmur will be well marked or not in any particular case of dilatation of the right ventricle.

1. The factor of prime importance is the over-distension of the first part of the pulmonary artery, whereby instead of being cylindrical it becomes, during systole, more nearly globular, resembling an aneurysm, and consequently the blood within it develops eddies and the aneurysmal murmur which they cause.

2. Together with this first factor must be noted the alteration in the direction of the axis of the pulmonary artery, as compared with that of the ventricle, which tends to favour the production of eddies in the dilated artery.

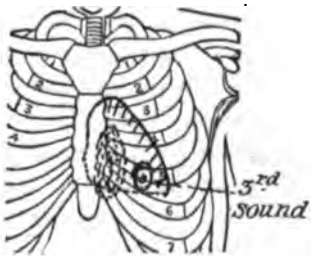


FIG. 81.

Kate B., aged nineteen. Some dilatation in second space and accentuation of pulmonary second sound, but no murmur. A tricuspid systolic and right ventricle third sound present.

3. The third main factor is the relative proportion between the size of the pulmonary orifice through which the blood enters as compared with the size of the dilated pulmonary artery itself.

For the production of eddies it is necessary that the orifice of entrance be smaller than the vessel into which the blood is entering. If the orifice of the pulmonary artery be normal in size and the artery beyond be abnormally distended during systole, the production of eddies and the resulting murmur will occur.

If, however, the orifice be dilated to the same extent as the artery, the vessel will retain its normal cylindrical form, in spite of its dilatation, and no murmur will result.

Of these three factors the second one need not be further discussed as there is, so far as I know, no evidence that variation in the loudness of the murmur is ever due to its variation—if such were possible.

The first factor—namely, the distension of the pulmonary artery—is an important one, and must be analysed into its component parts, for it depends upon the relationship between the internal pressure in the artery and the resisting power of the arterial wall.

Distension of the artery will not only occur with a normal or slightly raised intra-arterial pressure in conjunction with an arterial wall weakened by relaxation, but it may also occur in an artery with walls of normal tension if the blood pressure is sufficiently raised.

Also relaxation of the wall of the pulmonary artery will not suffice to produce a murmur if the blood pressure be so lowered —as a result of myocardial weakness—that it is insufficient to distend the relaxed vessel fully.

Upon the possible variations in the relationships of these two elements in the causation of the murmur a good many of the exceptional cases depend.

The third main factor also is variable where extreme degrees of dilatation are concerned, for it is almost certain that the faintness or absence of a pulmonary murmur which is noticeable in extreme cases of dilatation is due to the fact that the orifice of the pulmonary artery partakes in the enlargement of the adjacent part of the right ventricle.

This question will be taken up rather more in detail later on, and in the meantime this factor can probably be left out of consideration when dealing with the ordinary cases which are usually met with.

In the ordinary cases we meet with, the agencies producing the murmur are a relaxation of the elastic wall of the pulmonary artery owing to its origin being approximated to its termination by the upward dilatation of the right ventricle, combined with a blood pressure in the artery of normal amount and often distinctly in excess of the normal.

Now the loudness of the murmur depends upon the degree in which these two things are present.

Given a constant blood pressure, the greater the relaxation the greater the murmur; or given a constant degree of relaxation, the greater the blood pressure, the greater the murmur. The ready variability of the murmur can be understood when it is remembered that increase in the intraventricular pressure increases not only the relaxation of the artery by dilating the ventricle, but also increases the amount of the distension of the relaxed artery, and, in addition, the force by which the murmur-producing eddies are caused in the dilated artery.

As, however, the blood pressure does not as a rule vary very much in these cases, as far as its murmur-producing power is concerned, provided that temporary variations by recent exertion and excitement are eliminated, it may be left out of consideration in the majority of the cases met with. Therefore, when dealing with an ordinary series of cases it can be asserted that the loudness of the pulmonary artery murmur will depend upon the degree of relaxation of the vessel, and this is directly proportional to the degree in which the level of the pulmonary valves are raised by the upward dilatation of the ventricle.

This statement is borne out by the table at p. 264, when it is seen that the average loudness of the murmur gradually rises as the amount of dilatation increases until the dilatation reaches the somewhat extreme amount of two inches or so, when variation of the third main factor begins to interfere with the loudness of the murmur.

What has just been said may be stated in the following series of propositions:—

1. When the arterial wall is relaxed, as above described, the murmur will be faint or absent if the blood pressure be too low (owing to cardiac weakness) to distend the artery adequately.

In such a case, as the patient gets less anæmic and less breathless, it may sometimes be found that the murmur increases in loudness without any definite change in the amount of dilatation present, owing to the improvement in the blood pressure.

2. When the blood pressure is sufficient to distend the artery adequately, the loudness of the murmur will be distinctly proportional to the amount of relaxation and to the height of the blood pressure in the artery in each particular case.

3. Apart from relaxation of the arterial wall (such as we are speaking of), it is possible for a murmur to be produced by over-distension of the pulmonary artery provided that the right ventricle is strong enough to develop and resist an

intraventricular pressure which is in excess of the resisting power of the arterial wall.

4. When the other factors are constant, the loudness of the pulmonary systolic murmur will be proportional to the smallness of the pulmonary orifice in comparison with the size of the dilated artery, and conversely faintness or absence of the murmur will occur when the pulmonary orifice is so dilated that its size approaches that of the dilated artery.

I believe the pulmonary artery murmur in such a case as the following, where (although there was some yielding of the anterior wall of the ventricle) the murmur was louder in the erect than in the recumbent attitude, was due mainly to the action of the excessive blood pressure upon the elastic tissue of the arterial wall, and was therefore louder with the somewhat more strongly acting heart which is associated with the erect posture.

Case showing a pulmonary systolic murmur due mainly to excessive blood pressure in the pulmonary artery :—

The patient was a young man aged twenty, who was seen in December 1891, complaining of breathlessness, which prevented him from working. There was no history of rheumatism or other ailment, and the exact cause and mode of onset of his heart trouble was not noted. On examination, the heart was found to be acting tumultuously, and was often irregular. It was found to be dilated upwards and to the left, and a systolic pulmonary murmur was present, but there was no other murmur to be heard.

Two days later the heart was still very excitable, and there was a loud, rough systolic murmur heard in the second interspace both to left and right of the sternum, and the pulmonary second sound was very much accentuated. There was no murmur to be heard at the apex, but the first sound was short and sharp. There was one inch of relative dullness in the second left interspace with well-marked pulsation. Both the pulmonary second sound and the pulmonary murmur were fainter when he was lying down than they were when he was standing up. A month later, after being back at work for a fortnight, the murmur was not audible when he was recumbent and the heart much less excitable. The patient was much better, although at first the return to work made his heart bad again. This case is apparently one of asthenia of a transient nature, mainly involving the left side of the heart—such as that sometimes due to influenza—and the right ventricular dilatation was secondary to the ineffective action of the left ventricle (*vide* p. 300).

The dilatation of the conus arteriosus and of the pulmonary artery being secondary to failure of the left ventricle might be expected to be lessened when the heart was, owing to the recumbent attitude, acting more quietly than when the patient was erect.

It is a pity that in this case no definite note was made as to the effect of change of attitude upon the cardiac dilatation, for there is little doubt that it would have supported the explanation here given by showing no increase in the recumbent attitude.

I have not the notes available of any similar case in which a pulmonary murmur existed in the absence of an upward dilatation of the ventricle; but such are not very infrequently met with in somewhat older patients than those here dealt with, and more especially in men.

We have now to deal more fully with the last of the three main factors, and to point out its importance in explaining some of the cases which at first seem atypical.

As already stated, it is essential for the production of a murmur in the dilated artery that the orifice of entrance be smaller than the internal diameter of the dilated artery, for if the pulmonary orifice be dilated equally with the artery no pulmonary murmur can be produced.

This is, I believe, the reason why in most of the extreme cases of this type of dilatation the murmur is faint and sometimes entirely absent.

I ought to be able to furnish pathological proof as to the size of the pulmonary orifice in these cases; but until a few months ago, they were to my mind supposed to be explicable by a much more doubtful theory which I have held (without due analysis as is too apt to be our wont in matters clinical) for the last twenty years.

A few years' observation on such cases might give some positive evidence on this point, although the return of the structures towards normal, after death, as the result of rigor mortis, and their own elasticity would have to be reckoned with.

On turning to the table on p. 264, it is noticeable that out of two observations where there were three inches or more of dullness in the second left interspace, in one there was a faint pulmonary murmur, and in the other only a moderately loud one. In these cases of extreme dilatation—especially in those where the pulmonary valves lie in the first interspace, or under the first rib, it is by no means infrequent to find no murmur at all. Such are the cases illustrated by figs. 82 to 85, and at p. 314.

GREAT DILATATION OF RIGHT VENTRICLE. PULMONARY MURMUR FAINT

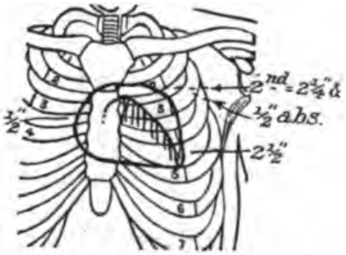


FIG. 82.

Ellen G., aged eighteen. Very considerable dilatation, but pulmonary murmur only audible in second left interspace.

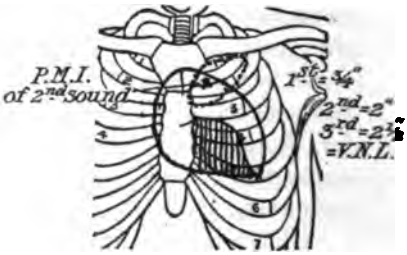


FIG. 83.

Sarah D., aged twenty-two. Great upward dilatation, but faint pulmonary murmur.

It is possible that in some cases of dilatation — and especially in those where dilatation is extreme—there may be another reason for the pulmonary murmur being less loud than might be expected: although the artery be relaxed, the pulmonary orifice not materially enlarged and the blood pressure sufficiently high. For in some such cases the artery may be prevented from becoming sufficiently globular to cause a loud murmur because of the support its walls are receiving from the adjacent structures. For instance, until recently, I supposed the absence of murmur which I have more than once noted in cases of pneumonia with great upward dilatation of the right ventricle, might be explained by the support afforded to the pulmonary artery by adjacent consolidation of the lung (*vide* p. 224). I now, however, think it more likely that these cases are explicable by the more recent theory of dilatation of the pulmonary orifice.

Again, in severe cases with a not too high blood pressure, it is possible that the support afforded by the adjacent structures (the aorta, bronchi, &c.) may be sufficient materially to lessen the amount of dilatation of the artery and so moderate the loudness of the murmur that would otherwise occur. In cases where the pulmonary valves lie at the upper border of the first rib, the extreme upward increase of the right ventricle must greatly limit the space available for any dilatation of the pulmonary artery; and it is possible that the external support thus afforded must be recognised as hindering the dilatation of the artery upon which the murmur depends. In these cases the anterior aspect of the pulmonary valves is often at least an inch away from the anterior chest wall, thus being displaced backwards as well as upwards; and the space available for the pulmonary artery, considering the close proximity of the transverse arch of the aorta, must be very small. On the other hand, the extreme elasticity of the fibrous tissues in adolescence must not be forgotten and the possibility of there being sufficient displacement of the aorta to allow of this dilatation.

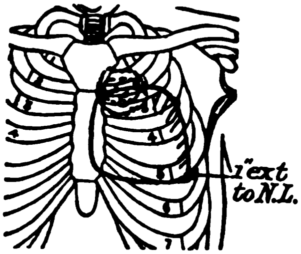


FIG. 84.

Emma B., aged seventeen. Very great upward dilatation, but faint murmur.

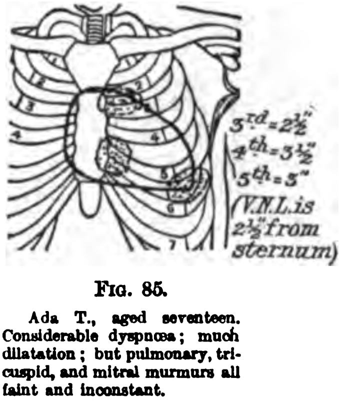


FIG. 85.

Ada T., aged seventeen. Considerable dyspnœa; much dilatation; but pulmonary, tricuspid, and mitral murmurs all faint and inconstant.

TRICUSPID AND PULMONARY MURMURS BOTH PRESENT
(THE AREA OF AUDITION OF THE MURMURS IS SHOWN BY DOTTED SHADING)

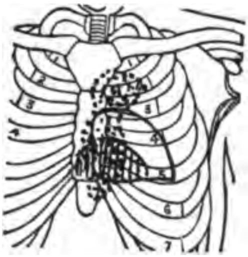


FIG. 86.

Annie B., aged fifteen.

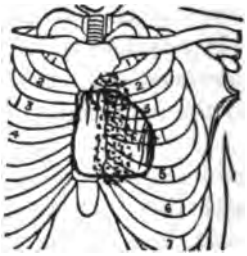


FIG. 87.

Emily B., aged nineteen.

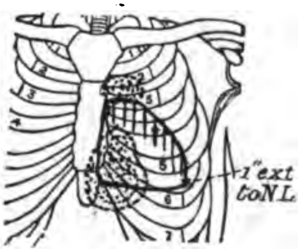


FIG. 88.

Adelina J., aged nineteen. Tricuspid systolic fainter if erect. Very faint pulmonary and no mitral murmur.

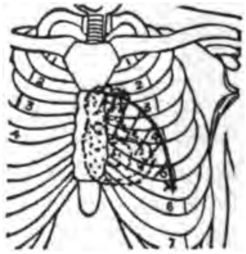


FIG. 89.

Amy H., aged nineteen. Some dyspnœa and faintness; pulmonary and tricuspid murmurs when recumbent, none when erect.

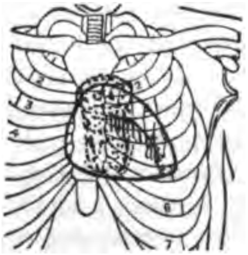


FIG. 90.

Emily N., aged twenty-two. Considerable dyspnœa. Well-marked pulmonary and tricuspid murmurs.

SUPERIOR VENA CAVA MURMUR, BUT NO TRICUSPID SYSTOLIC
(See also fig. 58.)

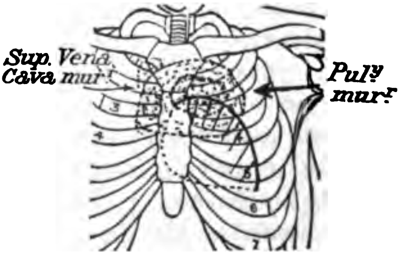


FIG. 91.

Eliza P., aged twenty. Loud pulmonary murmur; some dropsy of ankles. Tricuspid regurgitation, with superior vena cava murmur, but no tricuspid systolic.

In this connection, I may say that I have seen, in a case of sudden collapse of the left lung in a young woman, the heart shift from its normal position to such an extent that within a very few days its right border was in the left nipple line and the left border in the mid-axillary line—a sudden movement to the left of some 2 or 2½ inches in extent (*vide* p. 371).

Such a case as this seems to point to the fact that—in adolescence, at all events—we must not speak with too great certainty about the root of the lung as a 'fixed point,' owing its fixation to the spine by means of its fibrous tissue.

OTHER AUSCULTATORY PHENOMENA

Having now discussed the main phenomena associated with the heart failure which is apt to accompany anæmia in adolescence, it will be necessary to say a few words about some of the less important physical signs, and also to give some cases illustrating what has been said in the previous pages.

'BRUIT DE DIABLE.'—This well-known venous murmur which is so frequently heard in the neck in cases of anæmia is discussed in Essay VII, where a reprint is given of the writer's article on 'Dilatation of the Right Ventricle,' which appeared in the *Lancet* in 1894, and also at p. 15.

MURMURS DUE TO TRICUSPID REGURGITATION

Although definite dilatation of the heart to the right is by no means common in the heart failure of anæmia in adolescence (it occurred in 8·5 per cent. of the cases investigated), some signs of tricuspid regurgitation were noticeable somewhat more frequently—namely, in about 23 per cent. of the 178 cases, when definite notes as to its presence or absence were made (*vide* pp. 238, 355).

In one-fourth of the cases, the murmur was only faint. The accompanying figures (Nos. 86 to 90) show the shape of the area over which the murmur was audible.

A tricuspid regurgitation in these as in other cases may also show its presence by means of either a superior vena cava murmur or a venous valve murmur, with or without one or both of the other murmurs.

Fig. 91 shows a case where a well-marked superior vena cava murmur was unaccompanied by a tricuspid systolic murmur. There was a well-marked pulmonary murmur as well. Fig. 99 (p. 288) shows a case where there was a well-marked systolic murmur localised over the valves at the junction of the right internal jugular and innominate veins with a tricuspid and also probably a superior vena cava murmur.

CASES OF GREAT DILATATION SHOWING A FAINT OR ABSENT TRICUSPID SYSTOLIC MURMUR

Attention has been drawn (p. 265) to the fact that where there is an extreme amount of upward dilatation of the right ventricle, the pulmonary murmur, instead of being loud, is, as a rule, faint or absent.

The same phenomenon is also sometimes noticeable in the case of the murmur produced at the tricuspid orifice, and fig. 92 shows a case where great dilatation of the right ventricle to the right was unaccompanied by a well-marked tricuspid murmur.

As the tricuspid systolic murmur is more often present than absent in cases of extreme dilatation of the ventricle to the right, some different explanation must be sought from that which explains the absence of the pulmonary murmur under similar circumstances. Moreover, it must be remembered that the tricuspid systolic murmur is very often absent when there is only a moderate amount of tricuspid leakage. Therefore, the explanation of the feebleness of the murmur in the cases here given is probably the same as that of the absence of tricuspid murmur in other cases—whatever that explanation may be. In connection with these cases of considerable dilatation but no murmur, it may be of interest to give the following case of very considerable dilatation of the left ventricle, in which no mitral murmur was audible. It is hardly possible that in such cases the mitral valve still remains competent; but there is no means of proving that it is a case of mitral regurgitation without a murmur as can be done when such a condition occurs in connection with the right ventricle.

The patient was a girl aged twenty-two, suffering from a moderate amount of anæmia and considerable dyspnœa. There was some upward dilatation of the right ventricle, for there was an inch or so of relative dullness in the second left interspace with well-marked pulsation, but only a faint murmur. The apex was in the anterior axillary line in the fifth interspace; the impulse was heaving and forcible, and the first sound a little rough, but there was no true systolic murmur. There was no tricuspid murmur, nor was there any excess of right ventricular impulse in the fourth and fifth left interspaces (*vide* fig. 93 and also fig. 88).

Considerable Ventricular Dilatation, but no Regurgitant Murmur

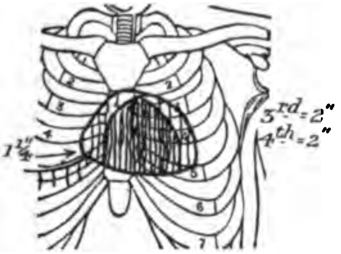


Fig. 92.

Eliza B., aged nineteen. Extreme dilatation; pulmonary murmur not loud. Slight true pulsation in neck veins, but no tricuspid murmur.

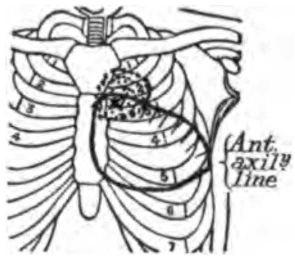


Fig. 93.

Minnie S., aged twenty-two. Extreme dilatation of left ventricle, but no mitral systolic murmur.

Pulmonary murmur present.

Arterial Compression Murmur. — This is a systolic murmur produced at the spot where the subclavian artery crosses the first rib and which is not infrequently heard in cases of anæmia in adolescence. It is discussed in Essay IV, which deals with the diagnostic difficulties associated with dilatation of the right ventricle (*vide* p. 226).

RIGHT VENTRICLE THIRD SOUND AND MID-DIASTOLIC MURMUR

These two physical signs which are by no means uncommon in right ventricular failure in anæmia are discussed in the essay before mentioned (p. 340), which contains a summary of the phenomena found in dilatation of the right ventricle.

Both the sounds here referred to are not at all loud, and they differ in this respect from the corresponding sounds on the left side of the heart which result from mitral stenosis. They are best heard when the binaural stethoscope is but lightly pressed against the chest wall.

The theory of air conduction by a binaural stethoscope necessitates a diaphragm whereby the sound vibrations of the chest wall are transmitted to the air in the stethoscope tube. The chest-piece, by imprisoning a portion of the skin, constitutes such a diaphragm, and it will, I think, be found that the most useful form of chest-piece is one so constructed that a variation in the firmness with which the stethoscope is applied causes a decided variation in the degree to which the imprisoned disk of skin is stretched.

In the case of the diastolic murmur and third sound, audible over the right ventricle in anæmic dilatation, the sound is so faint that a minimal amount of stretching of the 'skin diaphragm' is desirable.

The theory of the production of these sounds at the tricuspid orifice is quite simple if the expansion theory of the mid-diastolic mitral murmur, as discussed at p. 427, be accepted.

In the case of such hearts as these we are now describing, two factors are at work hindering the complete emptying of the auricle into the ventricle during the expansion phrase of the cardiac cycle.

Firstly, the heart muscle is apt to be lacking in vigour owing to malnutrition resulting from the anæmia, and its vigour of suction is likely to suffer in consequence.

Secondly, there is a tendency to auricular dilatation owing to the cardiac failure, and this will tend to ensure the prolongation of the flow of blood from the auricle into the ventricle, upon which the development of a third sound and mid-diastolic murmur depends. If the auricle holds more than its usual amount, the probability of blood passing through the valve, after the onset of the relaxation phase, is increased.

If carefuly looked for, one or other of these signs of incomplete aspiration by the ventricle will often be found in the cardiac failure of anæmia. In my series of cases, I have its presence noted only some nine times as occurring over the right ventricle and ten times over the left; but then I have only noted the absence of this sign about the same number of times; and if the presence of a third sound audible over the right ventricle were carefully looked for, it would be found, I believe, in quite a considerable proportion of the cases of well-marked anæmic dilatation of that ventricle. The following are three well-marked cases. In two of them a well-marked third sound occurred over the right ventricle, and in the third over the left ventricle. As the cases are also interesting from other points of view, they are given in some detail.

Cases Illustrating the Occurrence of a Right Ventricular Third Sound and Mid-diastolic Murmur

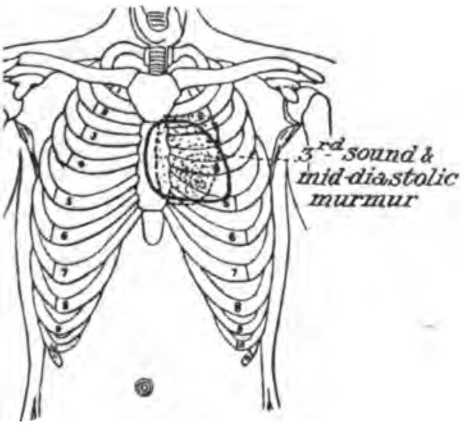


Fig. 94.

Clara B., aged nineteen. Showing area of audition of right ventricle third sound and mid-diastolic murmur.

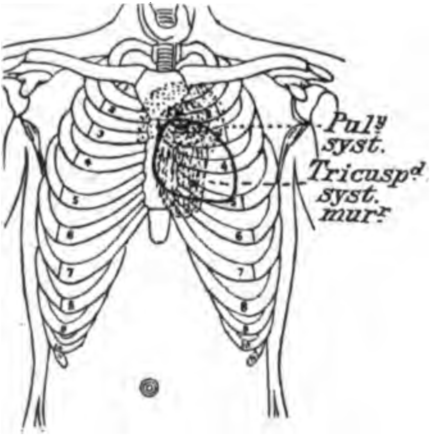


Fig. 95.

Clara B., aged nineteen. Showing area of audition of pulmonary and tricuspid systolic murmurs.

Clara B., aged nineteen, came to hospital suffering from marked anæmia, with breathlessness, weakness, and troublesome indigestion with emaciation. The heart was considerably enlarged upwards and to the left, there being relative cardiac dullness on full expiration for nearly 2 inches in the second interspace and to beyond the nipple line in the third (i.e. 3 inches from the sternal edge).

The apex was in the fourth space, three inches from the sternum. There was a loud, but decidedly localised, pulmonary systolic murmur, best heard over an area about two inches in diameter, and there was a well-marked reduplication of the second sound. There was also a tricuspid systolic murmur audible over, and just to the left of, the lower half of the sternum, and, in addition, a well-marked third sound followed by a 'mid-diastolic' murmur audible over the whole of the right ventricle, as shown in figs. 94 and 95.

In such a case as this it is easy to map out on the chest the exact position of the interventricular septum by studying carefully the area over which the third sound or mid-diastolic murmur is heard. The same is true of cases where the third sound arises in the left ventricle, for the sound is as a rule so feeble that the presence of the interventricular septum limits its audibility to the ventricle in which it arises.

In this patient the erect attitude caused a lessening in the size of the cardiac dullness and a total disappearance of all three murmurs and the third sound.

This patient rapidily improved under treatment without rest in bed, and in five weeks' time the heart had become normal in every respect.

In such a case as this with a well-marked true reduplication of the second sound, it is easy by means of a double chest-piece to the stethoscope (the so-called differential stethoscope) to demonstrate the presence of four cardiac sounds—a first sound, the two halves of the second sound, and the third sound.

The second case is that of Kate S., aged twenty-one, who was suffering from anæmia and dyspepsia with cardiac failure, when she came to hospital.

When recumbent, there was no relative dullness in the second interspace, but a faint pulmonary murmur—which was not well conducted—was audible. There were 2 inches of relative dullness in the third space (as against the normal 1½ inches), and the apex was under the fifth rib in the nipple line. The only other murmur present was a well-marked mid-diastolic one, heard over the point of maximum impulse of the right ventricle, which was situated in the fourth space half-way to the nipple line.

This was preceded by a well-marked third sound audible in this situation, and which was occasionally to be heard over the greater part of the right ventricle. The mid-diastolic murmur was also sometimes heard over this wide area. These areas are shown in fig. 96.

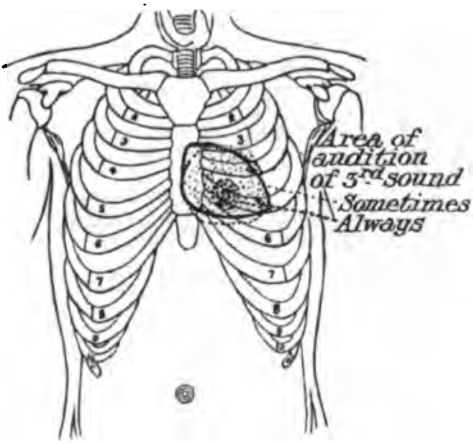


FIG. 96.

Kate S., aged twenty-one. Showing area of audition and P.M.I. of right ventricle third sound and tricuspid mid-diastolic murmur.

In the following case there was a third sound of left ventricular origin, but it was apparently functional in character and produced in the same way as the right ventricular one now being discussed. As the case is a very characteristic one of anæmic heart failure it will be dealt with somewhat fully.

The patient was a young woman of twenty-two, who came as out-patient to the hospital in March 1892, suffering from anæmia and so much breathlessness that she could barely manage to walk up a single flight of stairs without stopping to rest.

There was slight true pulsation in the veins of the neck and a very loud ' bruit de diable ' which, as usual, disappeared entirely when lying down. The heart was considerably enlarged, and there was 1½ inches of relative dullness with pulsation in the second left interspace, and in the third and fourth spaces the dullness and pulsation reached to the nipple line—i.e., 3 inches from the sternum. The apex was in the fifth space in the nipple line, and the right border was about ½ inch to the right of the sternum.

The pulmonary second sound was greatly accentuated, and the shock due to the closure of its valves could be clearly felt over an area $\frac{3}{4}$ inch in diameter, at the sternal end of the second left interspace. The second sound was distinctly reduplicated, although its two component parts were not completely separated from one another. The area over which the double sound was audible is marked in fig. 97. There was also a well-marked third sound, heard best over a small area 1 inch internal to the apex; but as its area of audition extended outwards towards the apex,

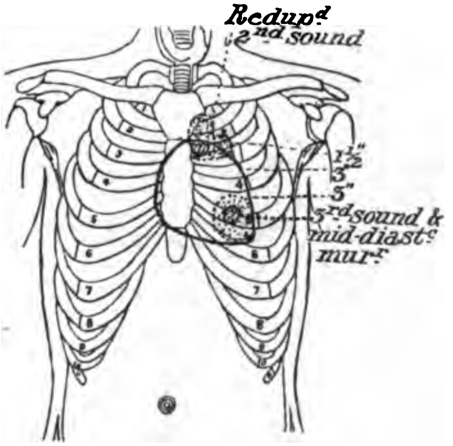


FIG. 97.

Alice K., aged twenty-two. Dilated heart and areas of audition of reduplicated second sound and of the third sound and mid-diastolic mitral murmur.

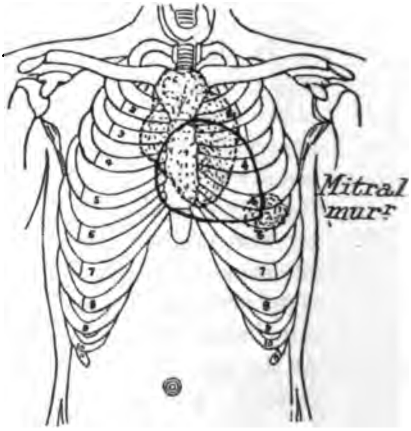


FIG. 98.

Alice K., aged twenty-two. Showing area of audition of systolic murmur at base of heart (probably a pulmonary tricuspid and a superior vena cava murmur), also a mitral systolic.

rather than inwards towards the sternum, its point of origin was almost certainly at the mitral valve rather than the tricuspid. The presence of a faint mitral systolic murmur, suggesting some dilatation of the left ventricle, tended to confirm this diagnosis. The third sound was not constantly present. In this case it was possible with a differential stethoscope to demonstrate the three diastolic sounds—namely, the two halves of the second, and the third sound. In addition, there was a systolic murmur audible over the pulmonary artery which appeared to be conducted downwards to the fifth rib and also across to the right of the sternum, and especially so into the second right interspace. [This last named must have been a superior vena cava murmur.]

Although this was noted as only one murmur, more careful study would—there is no doubt—have certainly shown that the portion below about the third rib was really a true tricuspid murmur, and that the portion to the right of the sternum in the second and adjacent interspaces was really a superior vena cava murmur. Change of position, holding of the breath, and the effect of exertion would, by varying the loudness of one or other portion, have sufficed to differentiate the three murmurs the one from the other (*vide* fig. 98).

This case is of interest as confirming what has been said as to the changes due to consolidation of the fibrous tissues of the heart wall and pericardium. It is a border-line case between the adolescent and adult types of dilatation. In addition to well-marked upward dilatation of the anterior wall of the right ventricle there was some general yielding as well, as was shown by the dilatation of the heart to the right and the evidence of tricuspid regurgitation which was given by the tricuspid murmur, the superior vena cava murmur, and the presence of some regurgitant pulsation in the veins of the neck. Further, the presence of some functional mitral regurgitation showed that the right ventricle was strong enough to allow an amount of exertion capable of slightly over-straining the left ventricle. Had the right ventricle been weaker—as would have been the case earlier in life—the girl would have had to give in sooner and would not have been able to continue her work until she overtaxed her left ventricle as well as her right.

This patient also rapidly improved, and the heart is noted as being nearly normal in five weeks' time.

CASES ILLUSTRATING CARDIAC DILATATION IN THE ANÆMIA OF ADOLESCENCE

It will now be well to emphasise what has already been said as to the character of the dilatation of the right ventricle in anæmia, and the murmurs which accompany that dilatation, by giving a series of cases showing the outline of the cardiac dullness and the areas over which the various murmurs were audible. Before doing so, however, it is necessary to say a few words in explanation of the extreme diversity in the shape of the cardiac dullness in the different illustrations, and also of the apparent want of correspondence between the amount of dilatation and the loudness of the murmur or the area over which it is heard.

The reason for the great variability of the physical signs here spoken of is to be found in the large number of factors that come into play in connection with the production of the dilatation and of the murmurs.

Case of Anæmia showing a High Apex Beat and a Normal Diaphragm

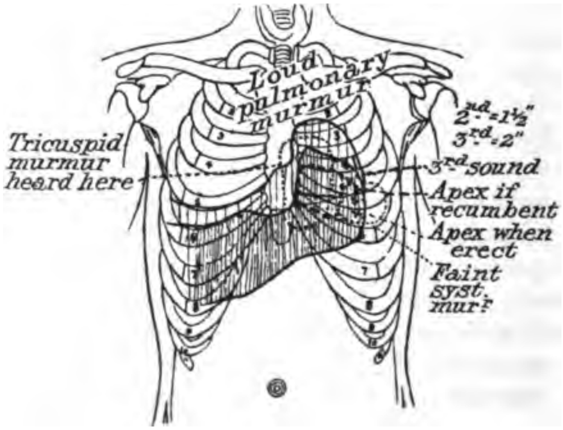


Fig. 99.

Vivian P., aged seventeen. Upward dilatation; loud pulmonary murmur, tricuspid murmur, and third sound, also faint mitral systolic murmur.

There was a venous valve murmur in the neck.

It might be well, therefore, in this connection to summarise the main factors, commencing with those which are most important.

The prime factors in causing the dilatation are the increased resistance in the pulmonary artery and the ability of the muscles of the right ventricle to develop sufficient power to overcome it. Both these are extremely variable, depending on the degree of anæmia and the degree to which the nutrition of the heart muscle suffers from the anæmia. Upon this last factor also depends the power which the right ventricle has of resisting the tendency to dilate, which is the natural consequence of the increased resistance in the lungs. Now the adequate filling of the left ventricle will depend upon the success which the right ventricle attains in its effort to pass the anæmic blood through the lungs, and there is every reason to believe that the apex of the heart is more displaced upwards where the ventricle is badly filled than when it is of normal size. This, then, is a factor which causes a most striking change in the shape of the cardiac area.

Again, a similar change to this last will result in those cases where the amount of malnutrition or the cardiac weakness is sufficient to cause a rise in the diaphragm owing to a lessening of the total volume of the blood in circulation.

The change in the outline of the cardiac dullness which results from this cause is well shown in the first two cases which follow. Compare figs. 99 and 100.

A Case of Anæmia showing a High Apex Beat with a High Diaphragm

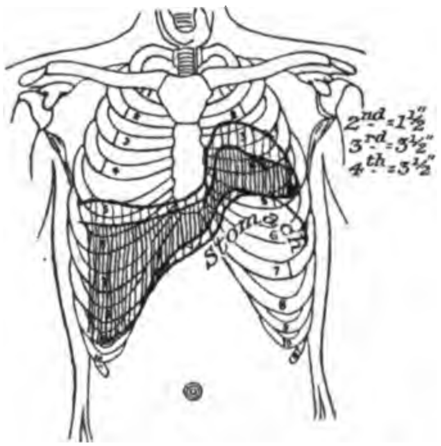


Fig. 100.

Florence P., aged eighteen, October 19, 1911. Marked dilatation of right ventricle upwards; high diaphragm, and moderate pulmonary murmur.

Variations in the Shape of the Cardiac Dullness

The shape assumed by the cardiac dullness in this type of dilatation is well shown by the figures given; but it may be well to say a word or two as to the frequency of the various types.

The most usual is where there is a moderate amount of outward dilatation proportional to the amount of pure upward increase, thus giving the heart's dullness an evenly rounded contour, such as is shown in figs. 66, 69, 94, &c.

Sometimes there is only upward dilatation present and without any other alteration in the normal shape of cardiac area (*vide* fig. 81).

Occasionally, a well-marked amount of upward dilatation is accompanied by a good deal of displacement upwards of the apex, but without any dilatation in the third left interspace—thus giving an irregular outline to the cardiac dullness, as is shown in figs. 101 and 103.

In this instance the conus arteriosus alone appears to be dilated. In such cases as this, it is apparently muscular feebleness which explains the absence of any increase outwards in the third space, because dilatation is apt to occur here also as the patient improves.

Another type which is sometimes seen is one characterised by broadening of the cardiac dullness in the third left interspace without any increase upwards (*vide* fig. 107).

The statistics as to age in relation to the amount of dilatation shows that this is more apt to occur in older than in younger patients (*vide* following table).

Average age of 153 cases of anæmic dilatation	19·3 years.
73 cases with 1 inch or less of dilatation in the second interspace	19·4 „
46 cases with 1¼ to 1¾ inches of dilatation in the second interspace	18·9 „
15 cases with 2 to 3 inches or more of dilatation in the second interspace	18·9 „
20 cases with no dullness in the second interspace, but dilatation in the third	20·3 „

These statistics, therefore, support what has been already said (pp. 31, 33) as to the greater tendency to upward dilatation in younger patients.

Two Cases of Raised Apex Beat with High and Normal Diaphragm Respectively

The following are two ordinary cases of anæmia, showing an ordinary type of upward dilatation of the ventricle, but in the one case the diaphragm was high and in the other normal.

Nevertheless, in both, the apex of the heart was in the fourth interspace when the patient lay down. When standing, however, there was a difference : for the low diaphragm of the first patient allowed the apex to fall into the fifth interspace when in the erect attitude ; the high diaphragm of the second patient, on the other hand, prevented any descent of the apex.

The first case was that of a girl, Vivian P., aged seventeen, who was suffering from anæmia and breathlessness. There was fullness of the neck veins ; a loud ‘ bruit de diable,’ also false pulsation and some true pulsation in them. There was considerable upward dilatation of the right ventricle, giving 1½ inches of relative dullness on ordinary respiration, with pulsation, in the second interspace, and 2 inches in the third. The apex beat was in the fourth interspace, nearly 3 inches from the sternum, but dropped into the fifth interspace when the patient stood up. There was a little absolute dullness in the third interspace. There was no increase of the cardiac area to the right. The liver was normal (as shown in fig. 99), both as regards the right and the left lobes.

Another interesting point about this patient was that in spite of her age being only seventeen, there was well-marked evidence of tricuspid regurgitation, although there were no signs of any dilatation of the right ventricle towards the right. There was a faint tricuspid systolic murmur audible over the area shown. There was also a third sound of right ventricular origin audible over the area marked by dark dots in fig. 99.

There was some true regurgitant pulsation in the neck, and a very loud systolic murmur and palpable thrill observable up to the angle of the jaw. In addition, there was the false pulsation and ‘ bruit de diable ’ usually noticeable in anæmic heart failure, and an ordinary pulmonary systolic murmur. The systolic thrill and murmur in the neck were of great interest, for they were not observable as loudly over the heart or in the aortic area. Neither were they to be found along the line of the subclavians, as is the case with an arterial compression murmur (*vide* p. 266).

The point of maximal loudness of this murmur—namely, just above the clavicle—and its direction of conduction up to the angle of the jaw, together with the fact that it was *not* loudly audible also in the subclavian region, made it certain that it must be produced in the internal jugular vein.

The true systolic pulsation in the neck veins showed the presence of a considerable amount of tricuspid regurgitation into them, and the cause of the murmur must have been this blood forcing its way past the valves which guard the lower end of the internal jugular vein (*vide* p. 219).

The second case was that of a girl, Florence P., aged eighteen, suffering from dyspeptic symptoms with breathlessness and only a moderate amount of anæmia.

The chief interest in connection with the case was that the breathlessness was associated with a high diaphragm on the left side, and the suggestion of defective circulation which this sign afforded was confirmed by the fact that the pulmonary murmur was not as loud as might be expected from the amount of cardiac dullness and pulsation in the second and third left interspaces; for in the latter space the pulsation could be felt nearly to the anterior axillary line, and there were 3½ inches of relative dullness. There was, however, a loud pulmonary second sound, and the right ventricle first sound was louder than normal.

The apex beat was in the fourth interspace, nearly 3½ inches from the sternum.

There was some reflex disturbance of the heart and circulation from a very irritable colon, and the hands were apt to go blue and congested and the heart's action was at times too excited.

Nevertheless, the case shows well a common type of anæmic dilatation of the right ventricle associated with a high diaphragm (fig. 100).

DILATATION mainly involving THE CONUS ARTERIOSUS.—In some cases, we find that while there is well-marked dilatation of the heart upwards into the second interspace there is no lateral broadening of the cardiac area in the third interspace. In some of these cases the outline given of the cardiac area retains more or less its normal shape. In others, although there is no dilatation in the third interspace there is in the fourth, often accompanied by—or due to—the rise of the apex of the heart from its normal situation into this interspace. This type of dilatation gives the somewhat striking outline shown in the figures illustrating the following cases.

The explanation of this type of dilatation must be found in the supposition that the dilatation only involves the conus arteriosus and not the remainder of the right ventricle, and it is confirmatory of the modern view that this part of the ventricle has activities and functions somewhat independent of the rest of the muscular wall.

Another point of interest which is brought out by the following clinical histories is that, where this type of dilatation occurs, it is sometimes found to give place to the ordinary type of dilatation as the patient improves, thus suggesting that (owing to muscular feebleness) the increased resistance in the pulmonary artery only caused dilatation of the conus arteriosus, whereas the return of a good measure of muscular strength caused a general dilatation of the anterior wall of the usual type. A more careful study of such cases than I have had the opportunity of making will be necessary to clear up the theory of their origin fully.

CASE OF ANÆMIA ILLUSTRATING DILATATION WHICH MAINLY INVOLVES THE CONUS ARTERIOSUS OF THE RIGHT VENTRICLE

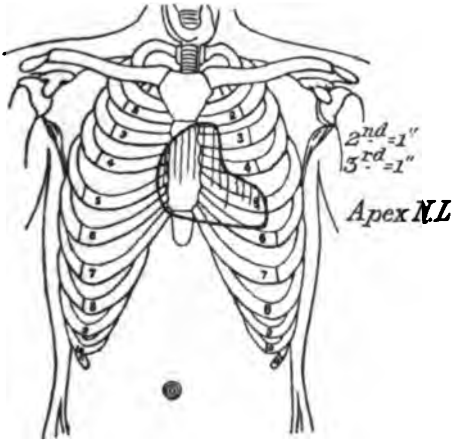


FIG. 101.

Theresa M., aged twenty-three, March 12, 1891. Showing dilatation mainly involving the conus arteriosus. There was a pulmonary murmur and a slight tricuspid.

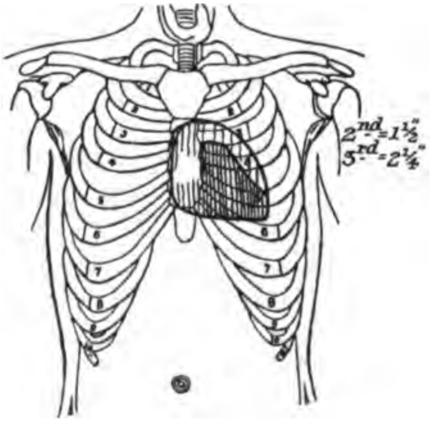


FIG. 102.

Theresa M., April 9, 1891. Much stronger, but more dilatation. Pulmonary murmur less loud. Increase of cardiac vigour had caused increased dilatation.

The first case is that of Theresa M., aged twenty-three, who came to hospital March 12, 1891, suffering from so much dyspnœa that she had to rest two or three times on going up a single flight of stairs, and could not walk a mile on the level.

There was but little anæmia and the patient was well nourished. The heart was as shown in figs. 101 and 102, and in the following table of measurements.

Relative cardaic dullness	March 12, 1891	April 9, 1891	April 30, 1891
	Inches	Inches	Inches
To left of sternum—			
2nd space . . .	1	1½	0
3rd ,, . . .	1	2¼	0
4th ,, . . .	(2½)	3	—
5th ,, . . .	3	3	3
To right of sternum . .	¾	Sternal edge	0

There was a moderately loud pulmonary systolic murmur on lying down, also audible over the sternum and to the right of it, and also a slight tricuspid murmur and some false but no true pulsation in the neck veins. There was no murmur at the apex. A month later when she was much better and able to manage one flight of stairs easily, or walk five miles on the level, the heart, instead of being smaller, was found to be more dilated, except that now there was scarcely any relative dullness to the right of the sternum, and the tricuspid murmur had gone. In spite of the increased amount of upward dilatation and the increased strength of the heart, the pulmonary murmur was less loud and was now not at all well marked. Three weeks later, the heart had returned to the normal size, but there was still a slight pulmonary murmur to be heard when she first lay down, and there was still some dyspnœa on walking uphill or upstairs. In this case (as in the next one to be referred to) the lessening of the pulmonary murmur must have been due to a dilatation of the orifice of the pulmonary artery when the increased upward dilatation took place.

The next case is one of considerable interest, and will be therefore dealt with rather more fully.

The following case is of interest from several points of view: Lizzie G., aged fifteen. Seen first in March 1891, suffering from anæmia, palpitation, and dyspnœa on exertion (*vide* fig. 103).

On examining the heart it was found to show the less usual type of upward dilatation of the right ventricle, which suggests

CASE SHOWING DILATATION OF THE RIGHT VENTRICLE WHICH MAINLY INVOLVES THE CONUS ARTERIOSUS

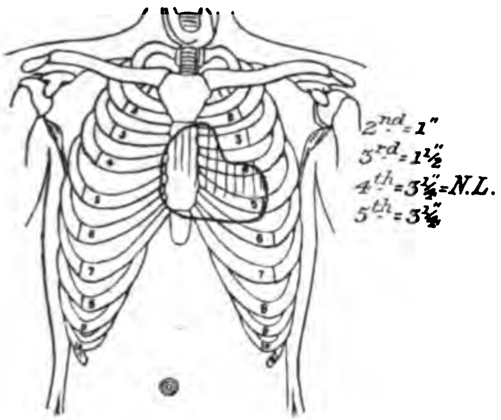


FIG. 103.

Lizzie G., aged fifteen, March 7, 1891. Dilatation mainly involving the conus arteriosus. Very loud pulmonary murmur. Great dyspnœa.

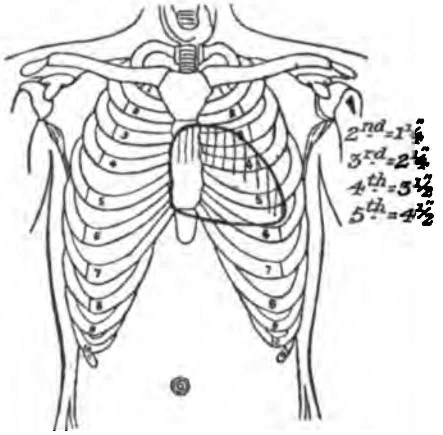


FIG. 104.

Lizzie G., April 4, 1891. Very much better, but more dilatation, and pulmonary murmur very faint.

Increased cardiac vigour had caused increased dilatation.

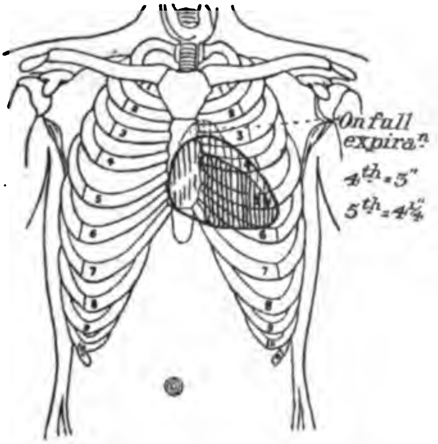


FIG. 105.

Lizzie G., May 2, 1891. Improvement has continued. The pulmonary murmur was now faint or absent. The dilatation had lessened.

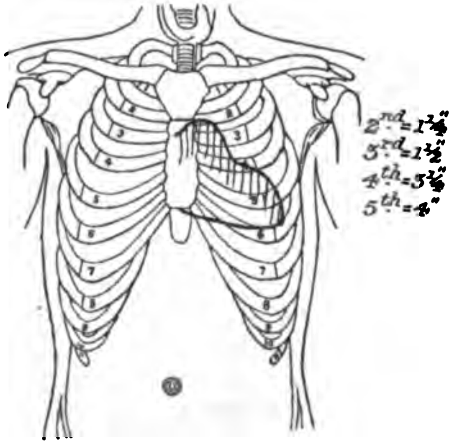


FIG. 106.

Lizzie G., November 21, 1891. Breathless and ill again. Faint pulmonary murmur. The original type of dilatation had recurred.

that the dilatation is limited to the conus arteriosus and does not involve the rest of the upper part of the ventricle; for, although there was one inch of relative cardiac dullness in the second interspace there was only the normal amount in the third and also in the fourth and fifth. There was a very loud pulmonary systolic murmur audible over the whole of the upper part of the chest and conducted along the arteries and audible even into the axillary arteries. There was no tricuspid murmur, and although there was a murmur at the apex, the possibility that it was only the pulmonary murmur could not be excluded.

She rapidly improved, and at her third visit a month later stated she was 'very much stronger,' for whereas a month ago she could only manage to walk a quarter of a mile, if she went very quietly—and even then would feel tired and overdone; she could now walk for an hour easily. Also, whereas a flight of stairs caused her to be breathless and exhausted, she could now do a flight quite easily (*vide* fig. 104).

With this history, it might be expected that the examination of the heart would show that it had returned to normal and that all signs of dilatation had disappeared. On the contrary, however, examination of the heart demonstrated the fact that it was decidedly more dilated than at her first visit (as comparison of figs. 103 and 104 will show), except in so far as the right margin of the relative cardiac dullness was concerned.

The accompanying table will best show the changes that took place in this patient's heart.

AMOUNT OF RELATIVE CARDIAC DULLNESS IN THE SEVERAL INTERSPACES

—	March 7, 1891	April 4, 1891	May 2, 1891	Nov. 21, 1891	Dec. 19, 1891	Jan. 30, 1892
	Inches	Inches	Inches	Inches	Inches	Inches
To left of sternum—						
2nd interspace	1	1¼	0 (1)	1¼	¾	0
3rd ,,	1½	2¼	1½	1½	1¼	1½
4th ,,	3¼	3½	3	3¼	3¼	2
5th ,,	3¼	4¼	4¼	4	3¼	2½
To right of sternum	½	Sternal edge	0	—	—	—
Pulmonary murmur	Very loud	Very faint or absent	Faint or absent	Faint	None	None
General condition	Very breathless	Much better	Much better	Breathless again	Much better	Very well

These two cases illustrate fairly well this special type of dilatation which is by no means a common one in anæmia.

It is remarkable that so great an increase in the dilatation of the right ventricle upwards should have been accompanied by so great an improvement in the patient's condition, and it strongly suggests the conclusion that, at first, the heart muscle was too weak to develop enough intraventricular pressure to cause dilatation, and that (as is the case in acute myocardial damage) dilatation of the heart was here a sign of returning strength.

The outlines of the cardiac area seem to show that this patient was always apt to have dilatation of the conus arteriosus out of proportion to the adjacent part of the ventricle. Another point of interest here is the lessening of the pulmonary murmur in spite of increasing upward dilatation and increasing strength. This was due in all probability to dilatation of the pulmonary orifice, as previously suggested. The fact that in this case a loud pulmonary murmur was never again noticeable is also in favour of this explanation. When seen on April 4, there was a slight tricuspid systolic murmur audible only over the right ventricle.

When examined, subsequently, on May 2 (*vide* fig. 105), there was, as is so often the case, well-marked reduplication of the second sound, and a note was made that the first portion was clearly of pulmonary origin and the second half of aortic, and that the pulmonary half was (as is usual) not audible at the apex. This observation can with care be often made in these cases of anæmia, thus confirming the fact that increased pressure in the pulmonary artery is the cause of the reduplication.

Another point of interest in this case is that she suffered for a time from optic neuritis as a result of her anæmia, and Dr. Lloyd Owen reported in May the presence of 'double optic neuritis with a medium amount of exudation and great vascularity.'

After some months, she came under treatment again for a slight return of her anæmia and of her dyspnœa; for although she could now walk a mile with fair ease, one flight of stairs would make her breathless. She had also some dyspepsia, which probably had much to do with her relapse. Fig. 106 and the above table show that the same type of dilatation occurred again, and they indicate the changes that took place in the heart until January 30, 1892, when it was quite normal and the patient quite well again.

Dilatation mainly to the Left.—It is not infrequent in cases of anæmia to find that the amount of dilatation outwards in the third and fourth left interspaces is in excess of that in an upward direction. Sometimes the whole of the dilatation is outwards; and if, at the same time, the lower border of the heart is raised, the cardiac dullness loses its ordinary shape and is very wide in proportion to its vertical diameter.

The following is the most extreme case of the kind of which I have records:—

Mary J. R., aged fifteen, but tall for her age, came to the hospital on November 14, 1891, complaining of shortness of breath, palpitation, and indigestion.

On examining the heart it was found that in the erect attitude there was no dilatation upwards or to the right, but that there was cardiac dullness and pulsation for 4 inches (i.e. nearly to the anterior axillary line) in both the third and the fourth left interspaces. The lower border of the heart was at the fifth rib.

On first lying down, ½ inch only of relative dullness appeared in the second interspace, but after a few minutes this increased till there was 1½ inches. On once again standing up, the dullness was slow in disappearing again.

The size of the heart at this visit and one paid a week later is best shown in the following table:—

Inter-space	November 14, 1891						November 21, 1891					
	Erect			Recumbent			Erect			Recumbent		
	Dullness		Pulsation	Dullness		Pulsation	Dullness		Pulsation	Dullness		Pulsation
	Relative	Absolute		Relative	Absolute		Relative	Absolute		Relative	Absolute	
	Inches	Inches		Inches	Inches		Inches	Inches		Inches	Inches	
2nd	0	0	0	1½	0	Yes	1¼	0	—	1½	0	—
3rd	4	0	+	4½	—	Yes	3	¾	—	4	¾	+
4th	4	3	faint	5	—	Yes	3¼	—	—	4	—	—
5th	—	—	—	No cardiac dullness or pulsation.								

The nipple line was 3 inches from the sternal edge and the anterior axillary line was 4½ inches from it.

The peculiar shape assumed by the cardiac dullness is shown in fig. 107.

When erect there was only faint pulsation in the fourth interspace, so the apex beat appeared to be in the third.

As regards the heart sounds there was a well-marked pulmonary systolic murmur heard over the second and third interspaces and conducted towards the neck, where it was lost in a loud murmur of subclavian origin. The pulmonary second sound was greatly accentuated and there was a 'bruit de diable' in the neck. At the second visit the pulmonary murmur was less loud, and there was occasionally a faint tricuspid murmur to be heard at the sternal end of the fourth left interspace.

Also at this visit there was a trace of relative dullness to the right of the sternum.

Fig. 108 shows another case where there was almost no dullness in the second interspace, but in the fourth it was ½ inch external to the nipple line. In this case, too, there was a slight tricuspid murmur.

Two Cases of Anæmia showing Broadening of the Cardiac Area

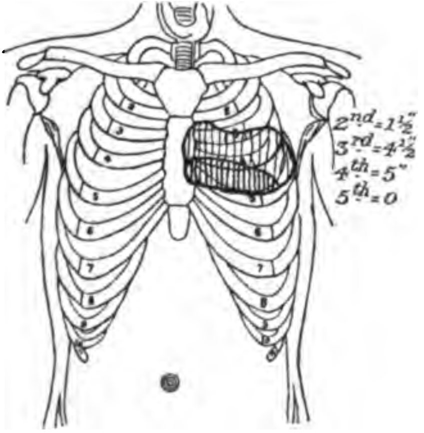


Fig. 107.

Mary J. B., aged fifteen, November 14, 1891. Extreme broadening of the cardiac dullness. Well marked pulmonary murmur.

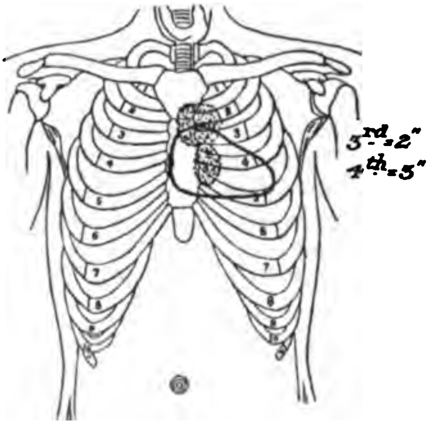


Fig. 108.

Alice G., aged sixteen. Great dyspnœa. Broad cardiac dullness with pulmonary and tricuspid murmur.

CASES SHOWING NO INCREASED LOUDNESS OF THE PULMONARY SYSTOLIC MURMUR IN THE RECUMBENT ATTITUDE

We must now speak of four cases where the pulmonary systolic murmur was not increased when the patient lay down, and see if it is possible to find a reasonable explanation why these do not conform to the general rule, for the test of a theory is its ability to explain the exceptions as well as the ordinary cases.

The first case is really not one of anæmia, but it is given here as an instance of a type of exception to the rule that we may occasionally expect to meet with. It has been referred to at p. 274.

The patient, who came to see me in December 1891, was a man aged twenty, suffering from breathlessness with occasional irregularity, and tumultuous action of the heart. There was no history of rheumatism, no mitral or aortic murmurs, and the main abnormality was dilatation of the right ventricle upwards and to the left, giving one inch of relative dullness and also pulsation in the second left interspace. There was also a well-marked pulmonary systolic murmur, which was noted as being loud and rough in character. There was great accentuation of the pulmonary second sound, and it was well heard in the second interspace to the right and the left of the sternum.

The interesting point was that the pulmonary systolic murmur, although quite loud and rough when he was standing, was quite faint or even absent when he lay down. It was also noticeable that the pulmonary second sound was less loud in the recumbent than in the erect attitude.

These facts give the key to the solution of the case. It is evident from the roughness of the murmur and the loudness of the pulmonary second sound that the wall of the right ventricle, although slightly dilated upwards, was strong enough to maintain a much higher intraventricular pressure than normal: the need for this higher pressure being found in failure of the left ventricle from influenza or other cause, and as to the nature of which I have no record. It appears, therefore, that the right ventricle in this case was sufficiently strong to cause an aneurysmal dilatation of the pulmonary artery, in which the factor of intra-arterial pressure played a far larger part than usual, and relaxation of the arterial wall a far smaller part. There was here no anæmia to cause muscular feebleness.

Now, as the murmur-producing dilatation seems in this case mainly due to the abnormally high blood pressure it is natural for the murmur to become less with the quieting of the cardiac action, which takes place on lying down. In this case the physiological fact that the heart beats more quietly when the patient lies down is confirmed by the lessening in the loudness of the pulmonary second sound, which was observable in this case when he assumed that attitude. We may from this case safely make the following generalisation: The increase in the loudness of the pulmonary systolic murmur, which is usually observable when the patient lies down, must not be expected in those cases where increase in the intra-arterial pressure plays a more important part in the production of the aneurysm-like dilatation than that which is played by relaxation of the arterial wall. Each of the four exceptional cases here being dealt with may well be explained by this same generalisation, although the records which I have are hardly full enough for a very confident statement. The cases were only hospital out-patients, and were seen twenty-one years ago. In one of them the patient was a woman of twenty-seven, in whom the ventricle walls would presumably be stronger and able to develop a higher blood pressure than in the majority of the cases dealt with in this essay. There was, moreover, a tricuspid systolic murmur present as well as the pulmonary one, thus showing that the thin part of the anterior wall of the ventricle *was* losing some of its adolescent weakness, and that the chamber was more inclined to dilate as a whole than simply upwards and to the left.

This same fact was also suggested by there being no increase of cardiac dullness in the second left interspace, although there was some in the third space—namely, nearly to the nipple line.

The systolic pulmonary murmur was faint, but was not increased on lying down, neither was the tricuspid murmur louder.

Presumably, in the case of the tricuspid murmur also, the lessening in the cardiac force, due to the recumbent position, fully neutralised the increased facility for regurgitation up into the superior vena cava, which would result from the absence of the force of gravitation (*vide* fig. 109).

Of the next case I have very few notes, but it is evidently one of the same nature as the last two. There was only about ½ inch of relative dullness in the second space, and it was nearly normal in the third, and the apex was low in the fourth space in the nipple line.

CASE OF ANÆMIA SHOWING NO INCREASE IN THE LOUDNESS OF THE PULMONARY MURMUR IN THE RECUMBENT ATTITUDE

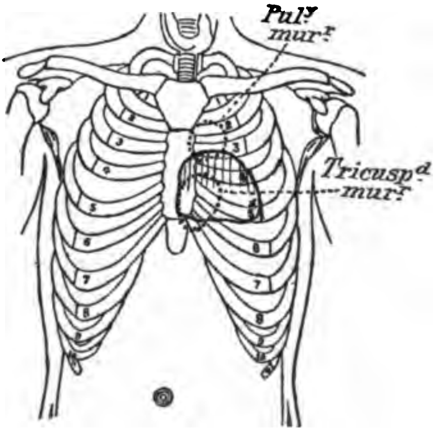


FIG. 109.

Alice A., aged twenty-seven. Pulmonary murmur not louder if recumbent.

There was a pulmonary systolic murmur localised to an area some two inches in diameter, and which was louder in the erect than in the recumbent attitude. The patient, a girl of nineteen, was anæmic, languid, and breathless (*vide* figs. 110 and 111).

The last case—though probably of the same nature as the other three—is not quite so clear, because a mitral murmur, which was present, was louder in the recumbent attitude. Unfortunately, I have no note of the loudness of the pulmonary second sound; but the fact that there was no increase in the amount of the upward dilatation of the right ventricle, when lying down, is in favour of the theory we are now advocating. In this case, too, there was only ½ inch of relative dullness in the second interspace, but the apex was in the fourth space ¾ inch external to the nipple line.

In the absence of fuller notes, a certain element of doubt must attach to this case. It is also possible that the additional loudness in the erect attitude may have been due to an increase in the loudness of the superior vena cava murmur rather than of the pulmonary systolic.

The following case is of interest as confirming what has previously been said with regard to heart failure without enlargement being due to a higher degree of muscular weakness than is the case when enlargement is present.

Case showing a Pulmonary Systolic Murmur Louder in the Erect than the Recumbent Attitude

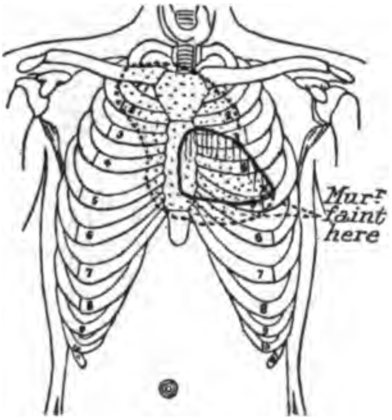


Fig. 110.

Edith E., aged nineteen. Showing cardiac area and area of murmurs when erect. The murmur on the right may be a superior vena cava murmur.

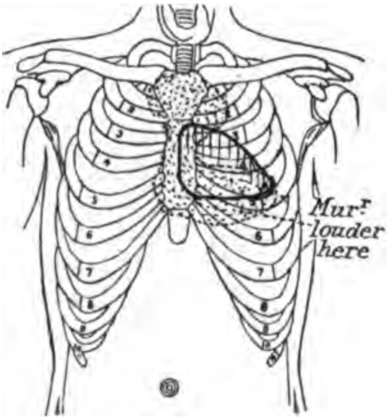


Fig. 111.

Edith E. Area of murmurs when recumbent. Pulmonary systolic less loud. Lessening of tricuspid murmur also.

In this case, however, the dilatation which occurred as soon as the heart began to get stronger may have been partly due to the fact that, owing to the patient not being confined to bed and not being under constant medical supervision, she overstrained her heart by too much exertion.

Had the patient been kept quieter it is probable that the dilatation would have been less; but, even if this were so, the case illustrates very well the diagnostic value of dilatation as pointing to good muscular strength as compared with the absence of dilatation which characterised the earlier stages of the patient's illness when there was evidently considerable muscular feebleness.

The case is as follows:—

Gertrude L., aged twenty-one, a watchchain-maker, came to the hospital September 15, 1891, complaining of frequent fainting attacks and great dyspnœa. She was very anæmic, and suffered from menorrhagia. The examination of the heart showed that there was some dilatation upwards and but little to the left and none to the right, as shown in fig. 112 and in the table, p. 306.

The apex was in the fourth interspace in the nipple line when the patient lay down, and under the lower border of the fifth rib when she was standing up. I have no doubt that had I known twenty years ago the importance of noting the level of the gastric resonance and the size of the liver, I should in this case have found that the former could be detected in the fifth interspace, and that the absolute dullness of the liver was greatly reduced in size: pointing to a degree of weakness of the cardiac muscle which would readily account for the feelings of faintness occurring several times a day—which was one of the chief symptoms of which she complained.

A Case of Anæmia showing Increased Loudness of the Pulmonary Murmur due to Increased Cardiac Vigour

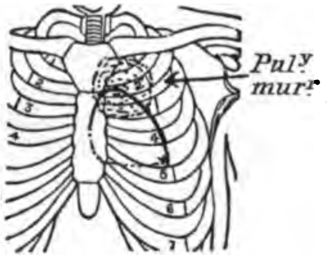


Fig. 112.

Gertrude L., aged twenty-one, September 15, 1891. Faintness and great dyspnœa.

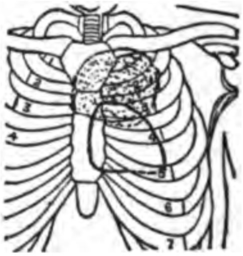


Fig. 113.

Gertrude L., October 24. No upward dilatation. Pulmonary murmur louder. Much better.

At this time (September 15) there was a distinct pulmonary murmur localised to an area a couple of inches or so in diameter with its centre over the situation of the pulmonary valves. She improved under treatment, and on October 24, was decidedly better, although she still had occasional attacks of fainting. Her palpitation was much less, and she could now go up two flights of stairs without having to stop, whereas she could not manage a single flight without considerable dyspnœa before September 15.

EXTREME DILATATION AND ABSENCE OF PULMONARY MURMUR

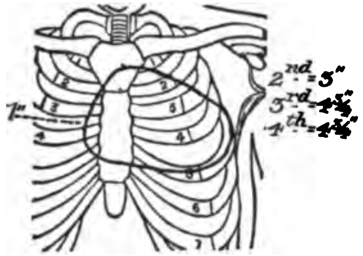


FIG. 114.

Gertrude L., December 5, 1891. Very greatly improved, but heart greatly dilated; pulmonary murmur *not* loud.

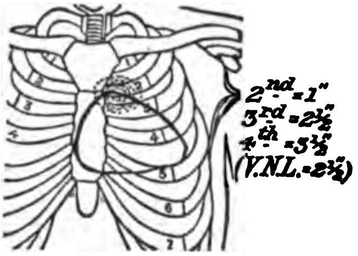


FIG. 115.

Gertrude L., January 2, 1892. Very much better.

This improvement was associated with a slightly lessened upward dilatation of the right ventricle, for there was now no dullness in the second left interspace as against an inch or more which was noticeable at the first visit (*vide* fig. 113).

The increased muscular power of the heart was shown not only by a lessening in the amount of upward dilatation, but also by an increase in the loudness of the pulmonary murmur: showing that in this case the lessened relaxation of the pulmonary artery was more than counterbalanced by the increase in the distensile force which resulted from the improvement in the tone of the heart muscle. The increased cardiac strength was also accompanied by a lowering of the apex beat from the fourth to the fifth interspace, due presumably to a lowering of the diaphragm.

When next seen on December 5, there was a great improvement in the patient's condition; she could now easily go up two or three flights of stairs and walk well, and had little breathlessness except when suffering from nervous excitement.

Coupled with this great improvement it was remarkable to find what might be considered a great deterioration in the condition of the heart, for it was very greatly dilated, as will be seen by a glance at fig. 114 or at the accompanying table.

When she lay down, the relative cardiac dullness reached up to the first rib and extended out to beyond the nipple line in the second interspace and to the anterior axillary line in the third. There was also one inch of relative dullness to the right of the sternum in the third and fourth interspaces, and there was well-marked cardiac pulsation all over this area. In spite of the great upward dilatation now present, the pulmonary systolic murmur was not at all loud.

	September 15, 1891		October 24, 1891		December 5, 1891		January 2, 1892	
Interspace	Erect	Recumbent	Erect	Recumbent	Erect	Recumbent	Erect	Recumbent
	Inches	Inches	Inches	Inches	Inches	Inches	Inches	Inches
2nd		(1¼)		—	2¼	3		1
3rd		(2)		(1¾)	4½	4¾		2¼
4th		2½		2¼	3¼	3¾		3½
5th	2½	—	2¼	—	—	—		—
To right	Mid-sternum		Mid-sternum		—	1	Right edge of sternum	
Murmur	Faint	Well marked		Loud		Not loud	None	Faint
General condition	Breathless and faint		Dyspnœa much less. Still faint at times		Dyspnœa *much* less. General improvement			

This case illustrates very well the interaction of the three main factors to which the pulmonary systolic murmur is due.

At the first visit there was relaxation of the pulmonary artery with a normal pulmonary orifice; but the murmur was faint because the ventricle was too weak to supply distensile force enough to cause an aneurysmal dilatation. At the second visit, with lessened relaxation of the arterial wall, there was nevertheless a loud murmur because the distensile force was great.

At the third visit there was very great shortening of the artery and therefore great relaxation of its elastic wall, combined with a very strongly acting ventricle—conditions certain to produce great dilatation of the artery; but, nevertheless, the murmur was faint because the dilatation was presumably great enough to involve the pulmonary orifice and so to prevent the production of eddies in the dilated artery.

At her fourth visit, three weeks later, the cardiac dilatation had greatly lessened and the patient was much stronger (*vide* fig. 115).

ESSAY VI.—THE HEART IN OVERSTRAIN

UNDER this heading those cases of heart failure will be dealt with where excessive physical exertion is the primary factor in the cardiac failure in contradistinction to many of the cases, already dealt with, where disease played a more or less important part in the breakdown of the circulatory apparatus.

As this is a very simple cause of cardiac failure, the giving of a few cases will help to illustrate the part which simple overstrain plays in cases of a more complicated nature, such as those due to anæmia which have already been dealt with. Looking at the cases of overstrain here given, it will be evident that as a rule there is nothing special about the changes in the heart which are found in them, but that they conform very closely to the changes in the right and left ventricles already described under Heart Failure with Enlargement in Adolescence in Adult, and in Later Life, and also in the essay on the Condition of the Heart in Anæmia.

It has just been said that this question of the effect of ordinary physical overstrain upon the heart is a simple one in comparison with many of the problems which face us in heart failure; but even here we are met at the outset with a question which has to be answered—namely, 'When the heart is overtaxed by physical exertion, does the pulmonary or the systemic circulation suffer more from the overstrain, or are both equally overtaxed?' In endeavouring to answer this question, we must remember what has already been said as to the difference between the heart of the adolescent and that of the adult—namely, that in the adolescent the heart's power of resisting dilatation depends upon its muscular wall, whereas in adult life the fibrous tissue of the pericardium and of the heart plays the most important part, and that, therefore, in early life the thinner portions of the heart walls are apt to dilate before the thicker parts, whereas later in life this factor does not play anything like so important a part.

If this fact were not recognised, we might make a mistake in answering the question we are at present considering, and say that in adolescence and early adult life it is evident that overstrain affects the pulmonary circulation much more than the systemic, for it is the right ventricle alone that shows overaction and dilatation in early life, whereas in adult and later life the left ventricle appears to be affected by overstrain to about the same extent as the right, or possibly more.

This statement would be an incorrect answer to the question now considered. We know that the ease with which the thin muscular wall of the right ventricle dilates in early life protects the stronger left side from dilatation, and therefore it is very difficult to tell by studying such cases whether the burden of overstrain falls mostly on the systemic or the pulmonary circulation. In adult life, when we have reason to believe that the distensibility of the two sides of the heart is much more nearly equal, we find that, in overstrain, the two sides of the heart are much more likely to be equally affected. This is not a point to which the writer has given special attention, and therefore all that can be said here is that there does not appear to be any great disparity between the extra strain thrown upon the right heart owing to the call for more rapid oxidation of the blood, and that thrown upon the left owing to the increased arterial supply demanded by the muscles. Doubtless a careful comparison of a series of cases would show that overstrain, due to exertion which necessitated fixation of the chest muscles—such as lifting heavy weights or rowing—would throw more strain on the right ventricle than on the left, whereas rapid general movements of the body—such as those required for bicycling—might involve the left side more than the right. Bicycle racing, where fixation of the arms and chest muscles is necessary, would probably come under the former category.

The well-known fact that in athletes it is the breathing that fails first—as shown by the retracted lips and wide open nostrils—must again not be taken as evidence that running, &c.,

throws more strain on the lungs than on the systemic circulation, because athletics are mostly practised by young men, in whom the distensibility of the right ventricle protects the left from overstrain, and, therefore, even though the strain be wholly on the systemic circulation, the only symptom of an overtaxed left ventricle in such a heart will be embarrassment of the right ventricle. This would be an interesting subject for further study.

Before giving the series of illustrative cases it will be well to say a few words as to the meaning of the term 'overstrain.' The actual meaning of the term implies that a heart is taxed beyond its powers of resistance and suffers in consequence—but this meaning would include all forms of heart failure; for a weak heart can be as much overtaxed in carrying on the normal circulation as a normally strong heart would be in doing an amount of work in excess of the normal. For the sake of convenience, however, it is, I think, usual to limit the meaning of the term to what may be called *absolute* (as distinct from relative) overstrain—namely, where a heart with no evident signs of weakness fails through being overtaxed by an excess of work. In some of the cases here given there was some heart weakness present, but this will not detract from the value of the series as showing the type of heart failure found in overstrain—as apart from valvular disease, &c. In other words, I have illustrated this subject with cases of relative overstrain as well as with those of absolute overstrain.

The first few cases given will be those in young individuals, and it will be seen that the type of dilatation of the heart is practically the same as that described in the essay on Anæmia. The first case I shall give is an ordinary one of simple dilatation of the right ventricle in an otherwise quite healthy young man, who had somewhat overexerted himself. It is a type of case very frequently met with.

J. E. B., a young man aged twenty-one, with no anæmia and of thoroughly healthy appearance, came to me in July 1893, complaining of breathlessness and feeling his heart beat too much after exertion. There was a history of overstrain at the age of fifteen, when he felt very ill and weak after a very long hill-climb on a bicycle. Latterly, he has only had dyspnœa after excessive exercise—such as rowing in a race. A month ago, he was nearly unconscious for the last half-mile of a boat-race, and also for half an hour afterwards.

When the heart was examined, it was found to be acting excitedly, and the first sound was too forcible. There was an abnormal amount of pulsation in the third and second left interspaces, but only when in the recumbent attitude, and in the epigastrium only when erect. The radial pulse was fairly good, but there was some false venous pulsation in the neck, and the veins were too full. The cardiac apex was in the fifth interspace in the nipple line. Percussion gave the following results: In the erect attitude there was only 1½ inches of relative cardiac dullness in the third left interspace—i.e. a normal amount—but on lying down the cardiac dullness increased markedly upwards, and the relative dullness was found to extend in the third interspace to the nipple—i.e. 2½ inches and 1¼ inches of relative dullness appeared in the second left interspace. The position of the apex did not change at all, so this increase was not due to a mere upward displacement of the heart as a whole, as is sometimes the case. In the erect attitude the first sound in the pulmonary area was not quite pure, but there was not a distinct murmur. When lying, however, a very well-marked murmur appeared over the pulmonary artery, and was audible upwards as far as the clavicles and episternal notch and downwards nearly to the apex. It was not conducted into the vessels of the neck, nor was it heard loudly enough over the lower part of the cardiac area to suggest the presence of a tricuspid murmur as well as a pulmonary murmur. There was no evidence of dilatation of the right ventricle to the right (*vide* fig. 116).

UPWARD DILATATION OF THE HEART IN OVERSTRAIN

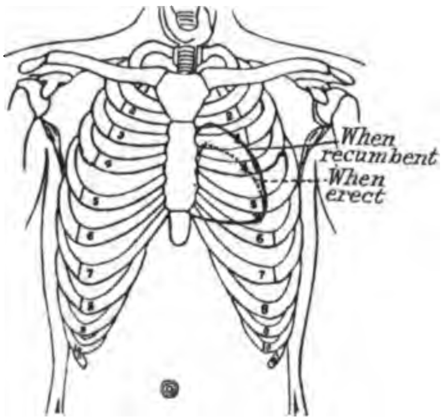


FIG. 116.

James E. B., aged twenty-one. Upward dilatation of right ventricle from overstrain. Dilatation increased on recumbency without alteration of the apex.

Here, then, was a fairly characteristic case of a right ventricle which had given way as a result of simple overstrain in a healthy young man. It is probable that the condition of the heart was not altogether the result of the recent boat-racing strain, but that the bicycling strain, five years previously, had left the heart unduly dilatable. This patient was examined again two years later, and although he considered himself quite well there was still a marked pulmonary murmur present, although there was almost no upward dilatation of the right ventricle. There was also a well-marked tricuspid murmur heard over the right ventricle in the fourth and fifth left interspaces and faintly to the right of the sternum.

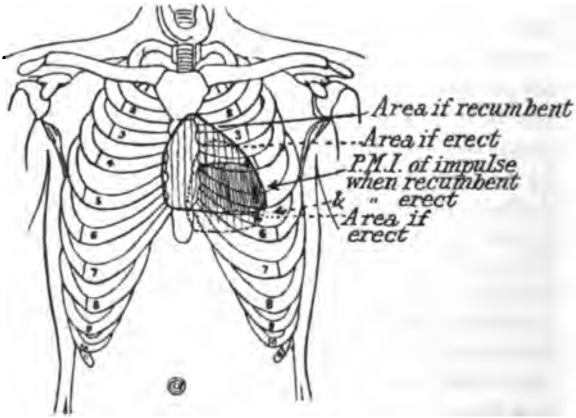


Fig. 117.

Jeffrey P., aged fourteen. Upward dilatation of the right ventricle from overstrain of a weak heart. Showing change due to recumbency.

The following is a good instance of the results of simple overstrain without anæmia in a boy of fourteen (*vide* fig. 117) :—

He was a well-developed and well-nourished lad—as shown by his height being 5 feet 1½ inches, and his weight 7 stone 10¼ pounds—but he had had an attack of rheumatism seven months previously, and had slight rheumatic pains occasionally since. There was no history of over-exertion, and the boy had not been to school since his illness; but no doubt the cardiac dilatation dated from shortly after his attack of rheumatism, as he was only kept in bed three weeks, although in it all his joints were involved and the temperature reached 102° F. on one or two occasions. The cause of the cardiac failure here was doubtless the softening and weakening effect of the rheumatic poison upon the heart, coupled with the undue strain caused by the boy being allowed to get up too soon, before the heart muscle had recovered from the effects of the rheumatic toxin. The case is, therefore, one of relative, not absolute, overstrain. The condition of the heart was as follows: There was about ½ inch of relative dullness in the second interspace, and a well-marked pulmonary murmur heard there when the patient was lying down, but when he sat up there was no dullness in the second interspace and no murmur. To the right, relative dullness was observable just to the right of the sternum in either attitude. There was some sternal dullness to the level of the third rib when recumbent, and to a less extent when erect. The apex was in the fourth interspace, just external to the nipple line (3 inches from the sternum) when recumbent. When erect, it dropped a little and could be felt in the fifth interspace, and some pulsation appeared at the same time in the epigastrium. In addition to these phenomena, a third sound was audible over a small area just internal to the apex when in the recumbent attitude, not when erect. This was of right ventricular origin.

Some six months later, the heart was in much the same condition, except that the apex was 4 inches from the sternum instead of 3 inches, and the apex beat could be felt in the fifth interspace when the boy was lying down. He was much stronger and better, but not so well as he would have been had he been made to take more rest and less exertion.

Another simple case of this same type of dilatation as the result of overstrain is that mentioned at p. 212.

The next case is a young man aged twenty, who came to see me in June 1893, suffering from breathlessness on exertion and attacks of fainting. He was decidedly anæmic, and was so breathless on exertion that he could not walk more than a mile or two quietly, and could not run at all. He had a little pain in the chest just above the left nipple if he overtaxed his heart. He was engaged in a flour-mill, and had strained his heart by dragging heavy bags of flour about. The neck veins were rather full with fluttering false pulsation in them. The heart was very greatly enlarged, and there was well-marked cardiac pulsation from the second to the fourth interspaces as far as the left nipple line, but none to the right of the sternum or in the epigastrium, though there was a little relative dullness in the fourth right interspace. The area of cardiac dullness was as follows: When in the erect attitude there was relative dullness in the second left interspace for 1¾ inches, and well-marked pulsation for 1 inch from the sternum. In the third and fourth there were 4 inches of relative dullness with pulsation over most of this area. The apex was in the fourth interspace, 4 inches from the sternum —i.e., more than 1 inch outside the nipple line. The maximal beat of the right ventricle was felt in the fourth interspace in the nipple line, 3 inches from the sternum. When this patient lay down, the resulting relaxation of the anterior wall of the right ventricle caused a marked upward increase in the amount of dullness, for the cardiac dullness was now well marked in the first interspace extending 1½ inches out from the sternum, and in the second interspace the cardiac dullness reached to the nipple line and there was an inch of absolute dullness (*vide* figs. 118 and 119).

UPWARD DILATATION OF THE HEART IN OVERSTRAIN

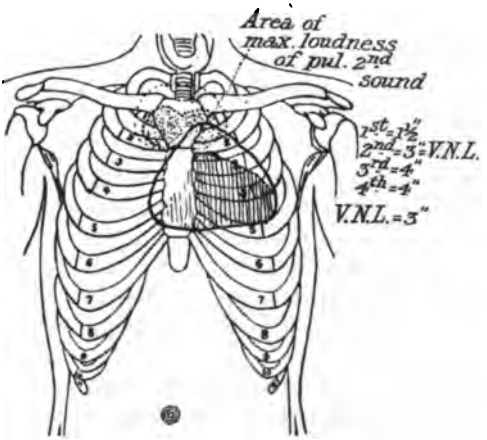


FIG. 118.

John M., aged twenty, June 19, 1893. Heart when recumbent showing extreme upward dilatation, but no pulmonary murmur.

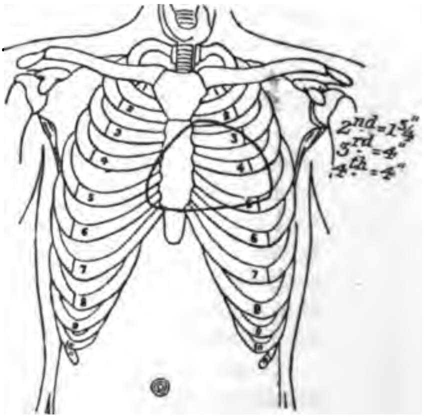


FIG. 119.

John M., June 19, 1893. Showing lessened amount of upward dilatation when erect, with no change in the situation of the apex.

The interesting point about this case—in addition to the somewhat extreme degree of upward dilatation of the right ventricle—was the complete absence of any pulmonary systolic murmur. The heart here was certainly strong enough to produce one, as was evidenced by the extreme amount of dilatation of the right ventricle, and also by an extremely loud pulmonary second sound. There was great upward displacement of the origin of the pulmonary artery, as was shown by the fact that the point of maximal loudness of the pulmonary second sound was in the first interspace, and, therefore, the pulmonary valves were above the level of the second rib instead of at the level of the third. Under such circumstances there ought to have been a very loud pulmonary systolic murmur, and the reason for there being none must be found in the fact that the pulmonary orifice was dilated as well as the pulmonary artery, and, therefore, no murmur was caused in the dilated artery.

In this case of overstrain, we see the typical appearances of a dilatation of the anterior wall of the right ventricle, such as is described in the essay on Anæmia.

Four months later, the heart still showed much dilatation upwards, for there was still 1½ inches of relative dullness and some pulsation in the second interspace, 2 inches in the third, and 3 inches in the fourth; but the amount of dullness was not materially increased when the patient lay down, although the amount of pulsation in the second interspace was increased. This showed an increase in the firmness and strength of the anterior wall of the right ventricle. The apex, too, was lower, and was felt in the fifth interspace when standing up, though it retreated behind the fifth rib when he lay down—showing a slight upward shift of the whole heart with the change of attitude. The point of maximal intensity of the pulmonary second sound was still in the first interspace, and there was still no pulmonary systolic murmur. The patient was much better and stronger.

The following case shows well how, in early life, the right ventricle dilates as a result of overstrain, although it is a case where the heart was not normal, but was so far weakened as a result of rheumatism that the intraventricular pressure necessary to carry on the circulation as the patient was lying in bed sufficed to cause the dilatation.

The patient, a youth of seventeen, was admitted to the General Hospital, August 12, 1892, suffering from a slight attack of rheumatism, which rapidly yielded to treatment with sodium salicylate. There was, on admission, a loud mitral systolic murmur with some dilatation of the left ventricle, the apex being just outside the nipple line. There was a slight amount of enlargement to the left in the third interspace, but no pulmonary murmur. Five days later, the pulse-rate dropped to forty, became very soft, the cardiac sounds lessened in loudness, and the slight amount of dilatation that was present on admission disappeared, the heart being of normal size. These signs all pointed to the onset of myocardial weakness. Two days later, with a return of cardiac strength, another type of dilatation appeared—namely, the upward enlargement of the right ventricle, as in the other cases noted. There was relative dullness for 1 inch from the sternum in the second left interspace, and for 2 inches in the third, and the apex was internal to the nipple line in the fifth interspace. There was visible and palpable pulsation in the second and third interspaces. The apical murmur was no longer present, but there was a well-marked pulmonary systolic murmur heard loudly both to right and left of the sternum in the second interspaces. There was also a faint tricuspid systolic murmur. For more than a week the dilatation and the murmurs remained well marked or slightly increased, and then a fortnight after its sudden onset, the dilatation began to subside, and the patient was discharged in a few weeks' time with a heart apparently normal.

This is a type of case which can extremely often be observed in rheumatism or other infective diseases, but it is very instructive as showing how the heart sometimes dilates with the return of strength which follows myocardial weakness.

OVERSTRAIN IN ADULT LIFE.—The following is an ordinary case of overstrain occurring in a healthy adult, and resulting in the ordinary adult type of dilatation of the right ventricle as well as of the left (*vide* figs. 120, 121, 122).

The case is as follows: John W., a man of about forty years of age, was taking a holiday in Switzerland, and on two occasions—once when walking up to St. Beatenberg, and once when walking up to Murren—had attacks of pain in the neck with some breathlessness. He found that the attack passed off quickly if he stopped and took deep breaths, but on the way up to Murren, he had to stop thus a dozen times or so. For some weeks after his return home, he noticed that he was gradually getting less able to hurry or to go upstairs quickly, and at the end of September, he was so alarmed at his inability (owing to breathlessness and a sense of powerlessness) to saw through a bit of deal 'only 3½ by 2½ inches' that he came to consult me.

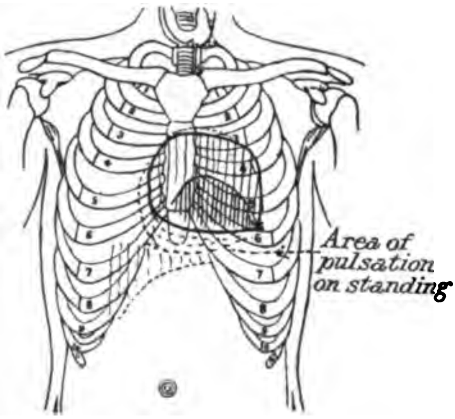


FIG. 120.

John W., aged forty, September 28, 1896. Showing area of cardiac dullness and pulsation. The dotted area shows epigastric pulsation only observable when erect.

On examination, he appeared well nourished and fairly healthy looking, but there was evident cardiac dilatation. The veins of the neck were rather full, and there was well-marked visible and palpable true pulsation in them. The radial pulse was regular—eighty to the minute—but small and of rather low tension. There was visible cardiac pulsation in the third,

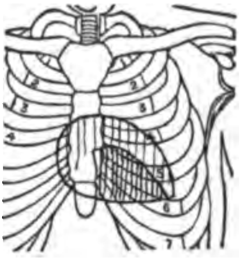


FIG. 121.

John W., October 6, 1896. After resting.

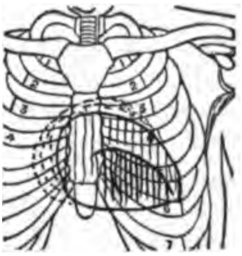


FIG. 122.

John W., October 20, 1896. Increase in the area of pulsation (shown by dots) after physical and mental exertion. Cardiac dullness much the same.

fourth, and fifth left interspaces and to the right of the sternum, over the area shown in fig. 120, and when he was standing up there was well-marked epigastric pulsation as well. The apex was low down in the fifth interspace in the nipple line. Its beat was feeble and wanting in vigour, and the left ventricle heart sounds also were short and wanting in tone. There was no murmur audible anywhere, and the right ventricular sounds were of better tone than the left, and the pulmonary second sound was rather accentuated.

This condition improved somewhat under treatment, but he would not take absolute rest, consequently the heart remained in an irritable condition, and dilated very easily as the result of physical and mental work, as may be seen by comparing fig. 121 with fig. 122. It was not until he remained a fortnight in bed that the heart returned to its normal size, and began really to regain its strength, but it was some months before he was able to do a full day's work.

The following is a case of overstrain in which, owing probably to relative weakness of the left ventricle, the resulting dilatation mainly involved the right side of the heart. The case is of interest because, in spite of marked fullness of the veins, some tricuspid regurgitation and upward dilatation of the right ventricle, accompanied by a pulmonary systolic murmur, there was no increase in the cardiac dullness to the right of the sternum, although there was some pulsation to be felt in that situation. Another point of interest is that the venous over-distension appears to have caused so much increase in the thoracic contents that the diaphragm assumed a lower level than normal, for the line of the liver dullness was an interspace too low. The apex beat also was in the sixth interspace instead of the fifth, when the patient stood up, and the pulsation of the right ventricle in the epigastrium was abnormally distinct when he was in the erect attitude.

The patient was a man, B. W., aged thirty-seven, who came to me in June 1911, complaining of breathlessness, irregular action of the heart, and some pain in the chest—apparently of cardiac origin. There was a history of his having strained his heart while bicycling eleven years ago. Since then, he had been well enough to play tennis and be a first-rate player, but he had to be careful about continuous exercise—such as running. There was a recent history of nervous overstrain, owing to difficulties in his work—he was manager of a brewery, and the badly ripened barley (due to the last season's wet summer) caused much trouble and anxiety. About two months prior to being seen by me, he had begun to get unduly nervous, the pulse at times running up to over one hundred, and the attack being accompanied by faintness. He then had an attack of acute indigestion — apparently a true gastralgia or a continuous transverse colon pain, which was not made worse by food or by exertion, and was relieved by rest and milk diet. While still on milk diet, he played in one week two long and closely fought tennis matches, for which he had not sufficient strength, and began to suffer from breathlessness, cardiac irregularity, and some cardiac pain over the apex of the heart. He was a healthy looking, well-built man. When standing, there was well-marked high pressure pulsations in the veins just above the clavicles, which could be felt to stop short at the venous valves. There was also some excessive arterial pulsation in the neck, but its type and its amount did not suggest aortic disease. The cardiac impulse could be felt too forcibly over the lower end of the sternum and in the epigastrium, also just to the left of the sternum in the fourth and fifth interspaces, and just to the right of the sternum under the fifth and sixth ribs. The right ventricular sounds were also loud and good, and there was a distinct, but localised, systolic tricuspid murmur, and also a well-marked pulmonary murmur, although there was no upward increase of the heart into the third and second left interspaces. On lying down the epigastric impulse nearly disappeared, the tricuspid systolic murmur became louder, and well-marked systolic pulsation was

Low Diaphragm in Case of Overstrained Heart

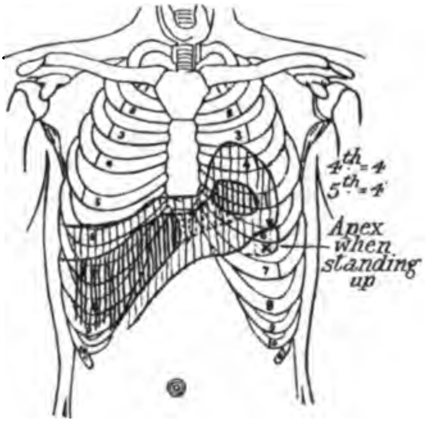


Fig. 123.

B. W., aged thirty-seven. Overstrain with feeble left ventricle and stronger and dilated right ventricle. Diaphragm low, probably from venous engorgement of lungs.

visible in the neck veins nearly to the angle of the jaw. The pulse was fairly strong and its tension too high. The sphygmomanometer showed a blood pressure of 160 mm. of mercury, and at 140 mm. the pulse became quite full and strong. The left ventricle was somewhat feeble, both as regards its impulse (which could be barely felt) and also its sounds, which were weak and accompanied by a faint mitral systolic murmur. The apex was too low, being in the sixth interspace when he was standing, and low in the fifth when he lay down. There was also some dilatation, for the outer border of the relative cardiac dullness was an inch external to the nipple line—4 inches from mid-sternum. The situation of the heart pointed to its being displaced downwards by lowness of the diaphragm (*vide* fig. 123).

The after-history of the case was uneventful, and the heart gradually regained its strength under proper treatment. Within a month the left border of the heart had come in 1 inch, the apex being in the nipple line instead of an inch external to it.

Overstrain in Later Life.—The following is a somewhat extreme case of dilatation, the result of continuous over-exertion, in a lady of eighty-one, whose nervous energy was in excess of the muscular power of her heart. I was asked to see her after a four hours' railway journey on account of some breathlessness and faintness.

She was rather stout, but was, on the whole, wonderfully active both in mind and body for her age, and I was surprised on examining the heart to find what an extreme degree of dilatation existed. There was cardiac dullness for more than 3 inches to the right of the middle line, and for 7 inches to the left, the apex of the heart being in the seventh interspace in the anterior axillary line. The transverse diameter of the heart in this case must, therefore, have been well over 10 inches, which showed extreme dilatation, considering that the patient was not (in spite of her stoutness) much, if at all, above the normal as regards height and build. In spite of this extreme dilatation the heart sounds were fairly loud and good, and there was no murmur present except the one described at p. 44 as the superior vena cava murmur. This murmur was only audible to the right of the middle line, up and down over (and just to the right of) the right border of the sternum from the neck to the fifth interspace. The size and shape of the heart is shown in the accompanying figure (fig. 124) which shows the extreme size that was observed. The heart in this patient had lost so little of its elasticity, that a few days of absolute rest in bed would reduce its size and the amount of pulsation considerably. For instance, after walking a dozen paces to and from the lift,

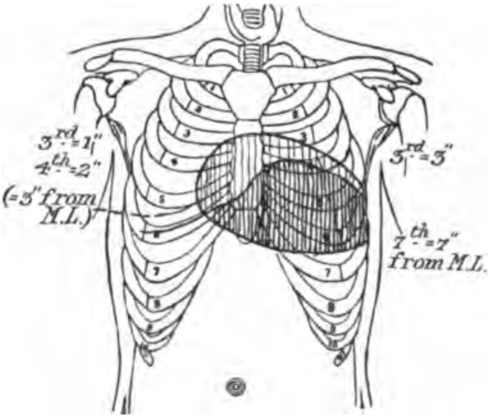


Fig. 124.

Mrs. W., aged eighty-one. Extreme dilatation of heart from over-activity. No murmur except a superior vena cava one.

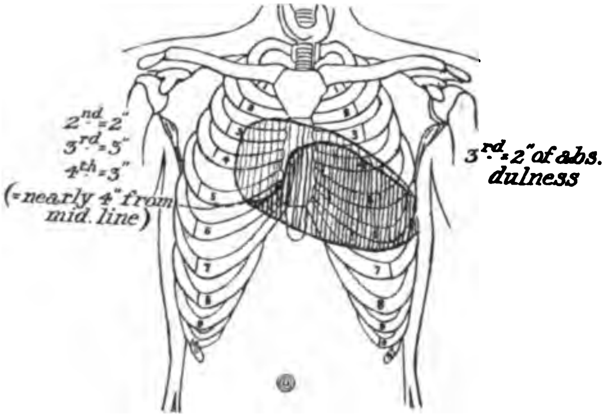


Fig. 125.

Mrs. W., aged eighty-one. Showing great increase in dilatation caused by a little more exertion than the heart would stand.

and spending the morning downstairs in a wheeled chair, the heart in the afternoon would show (as in the figure) 2 inches of relative dullness in the second right interspace, and nearly 3 inches to the right in the lower interspaces—i.e. nearly to the right nipple line (*vide* fig. 125). There would be pulsation to the right of the sternum, and the cardiac dullness would be nearly absolute just at the right border of the sternum. To the left there would be absolute cardiac dullness in the third interspace for 2 inches, and relative dullness to the left nipple line with well-marked pulsation. After a few days' rest, the heart's apex would come in half an inch or so, the pulsation and absolute dullness would be no longer noticeable in the third left interspace, and the dullness and pulsation to the right of the sternum would lessen.

If this patient had had proper advice years before and been willing to follow it, most, if not all, of this dilatation might have been prevented, for there was in all probability no valvular disease, although the absence of valvular murmurs could not be taken as certain evidence of this. It is not possible to say that there was no functional mitral regurgitation present. It is quite clear in this case—as shown by the presence of the superior vena cava murmur and pulsation of the veins in the neck—that, as might be expected, well-marked tricuspid regurgitation was present in spite of the absence of any valvular tricuspid regurgitant murmur. The same physical causes that prevented the development of a tricuspid regurgitant murmur (in spite of the presence of free regurgitation) may also have prevented the development of a mitral regurgitant murmur, in spite of the presence of free mitral regurgitation. In cases of extreme enlargement of the left ventricle, we do sometimes find (as in this case) that there is no systolic murmur audible, although the amount of dilatation makes it practically certain that the valve must be incompetent. Such a case is also recorded at p. 281.

EXTREME DILATATION IN ADOLESCENCE.—Finally, I will here give a case of overstrain of the right ventricle alone in a girl of sixteen, due to extensive inflammation of both lungs, the result of pyæmia (*vide* fig. 126).

A neglected otitis media was the cause of her pyæmia, and when I saw her, on October 24, 1898, three days after her admission to the surgical wards of the hospital, the heart was enormously dilated—as is shown in the accompanying diagram. There was pulsation, and 1 inch of relative dullness in the first left interspace. In the second interspace, the dullness and pulsation extended to beyond the left nipple line, and in the third and fourth interspaces to the anterior axillary line, some 5½ inches from the sternum, and absolute dullness reached up to the third rib. To the right there was relative dullness and pulsation for 1 inch to the right of the sternum in the third and fourth right interspaces, giving a total width of the cardiac area of more than 7 inches. There was a pulmonary systolic murmur heard over a small area in the first and second left interspaces, but no other murmur was to be heard. Next day, although the cardiac dullness remained the same to the left of the sternum, a pulmonary murmur was no longer audible, and the dilatation to the right of the sternum was less, being only ½ inch in width. The disappearance of the pulmonary murmur, coinciding with a lessening of the amount of dilatation of the heart to the right of the sternum, is confirmatory of the explanation given at p. 275 as to the absence of the pulmonary murmur in cases of extreme dilatation, for it suggests a slight gain in the efficiency of the right ventricle, and such a slight rise in the intraventricular pressure as might well have caused a dilatation of the pulmonary orifice, sufficient to cause the disappearance of the murmur.

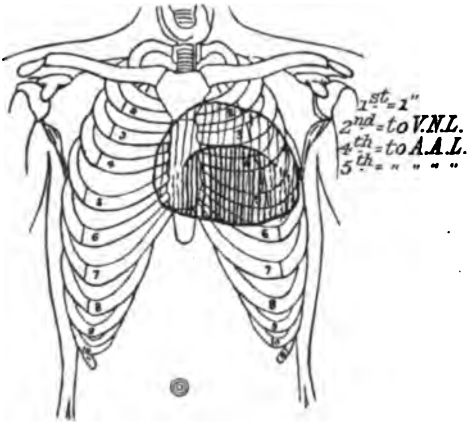


FIG. 126.

Extreme dilatation of heart in pyæmic pneumonia.

This is the case spoken of at p. 7 in which, at the post-mortem examination a day or two later, the heart was described as of normal size, but where a closer examination showed that the walls of the right ventricle and right auricle were both very thin and very distensible.

ANOMALOUS CASE OF OVERSTRAIN.—The following is a somewhat unusual and interesting case, because the overstrain caused heart weakness and tachycardia of a lasting description, and not simply dilatation as is usual. The cardiac injury was a result of the combined nervous and physical overstrain accompanying an accident, and it raises some interesting questions in connection with the pathology of tachycardia.

The accident was as follows:—

The patient (a man aged about thirty, a coal-miner by occupation) was sitting on the front of a full coal-tub, which was running down towards the shaft along a 10 feet wide 'road,' when it suddenly ran off the rails, and he had, by a violent effort, to jump off the tub sideways in order to save his life. The tub ran into the wall and knocked the roof-props down, and the roof of the passage fell in. The suddenness of his jump put his own lamp out, and he had to sit in darkness, hearing the roof falling in all round him, and expecting every moment to be crushed to death. He had to sit thus for ten minutes before he was rescued from his dangerous position.

Although the sudden effort of the jump for his life was not calculated to cause any severe cardiac damage, the prolongation of the state of strain by the nerve shock which followed it seems to have been sufficient to cause very severe damage. An additional reason for the severity of the cardiac overstrain may, I think, be found in the following considerations: The sudden violent effort caused a sudden violent action of the heart. Under ordinary circumstances this would have done no damage, as the heart would, in a few seconds, have returned to its normal mode of beating. The occurrence, however, of fear kept up the overaction of the heart for several minutes, and led to an amount of overstrain that did cause damage to the heart.

I first saw the man more than three years after the accident, and he was then incapacitated from work, because of palpitation, pain, and breathlessness. There was then no cardiac dilatation, and the physical signs suggested muscular weakness of the heart involving the left side more than the right. The pulse-rate was over 110 on slight exertion, but after a few minutes' rest would drop to 90. Slight exertion would cause the appearance of right ventricular pulsation to the left of the sternum, and it is, therefore, probable that both his pain and his breathlessness were due to the over-distension of the right ventricle, which resulted from its attempt to compensate for the inability of the left ventricle to meet the call for increased work during muscular activity.

An interesting question raised by this case is the following: What part did 'tachycardia' play in the cardiac disability of this patient? To make my meaning clear, I will give an illustration of a different kind. A lady was walking along a road where some boys are playing with a football. She was suddenly struck a severe blow on the side of the head by the football; moreover, as it was kicked from behind her, she could not see it coming, and, therefore, nervous shock due to sudden fright accompanied the physical shock of the blow. The result was that the sudden powerful contraction of the neck muscles, which was necessary to prevent damage to the cervical spine, instead of being, as might be expected, a *single* contraction, was perpetuated as a *clonus* of the neck muscles, and spasmodic torticollis resulted. I saw the patient a few weeks afterwards, and she was then a typical case of severe spasmodic torticollis. The habit of clonic spasm, started by the accident, had become firmly established, and it took many months of most assiduous exercises before the habit could be broken, and it was two years before the patient was reasonably free from clonic spasm of the neck muscles.

The question now arises: How far was the irregular action of the heart in the case of the miner due to the sudden and violent jump for life, and then the keeping up of the rapidity for ten minutes by the terror of that time of waiting in the dark with the roof of the passage continually falling in around him? It is probable that there was both physical strain with damage to some of the cardiac fibres combined with what may be called habit tachycardia. When he came under my care, three years after the accident, he was admitted into hospital, and kept quiet in bed without any treatment directed to the heart, except a little nickel sulphate. The heart's rate gradually came down, and after some weeks, when he was gradually allowed to resume a certain amount of activity, the tachycardia did not return. He gradually improved after this—finding that the amount of work he was able to do, without causing a return of the rapidity, steadily increased, but even after the lapse of a year the pain and rapidity would return as a result of too much digging in his garden, or of walking uphill, and anything approaching ordinary manual labour was still out of the question. A few notes as to his condition after the accident and on admission to hospital two years later may be of interest.

After the accident, he did not notice any pain in his chest for the first three days. Three days after the accident, he began to have pain in the precordial region, but, in spite of its gradually getting worse, he went on working for a month, when it got so bad that he had to give up. He describes the pain as starting in the left axilla about the sixth intercostal space, and shooting up to the shoulder. On reaching the shoulder, the pain would radiate out over the neck, giving a sensation like electricity. The pains would usually come on when he sat down after exertion rather than during its continuance. The pains were accompanied by dyspnœa. At the time of his admission to the General Hospital, three years after the accident, he was still liable to have shoots of pain as above described, lasting two or three minutes, if he overexerted himself in any way. These shoots of pain seemed to be accompanied by such forcible action of the heart that the patient himself could feel it thumping in the chest.

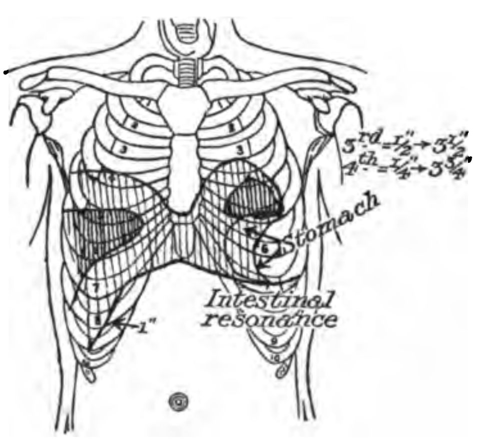
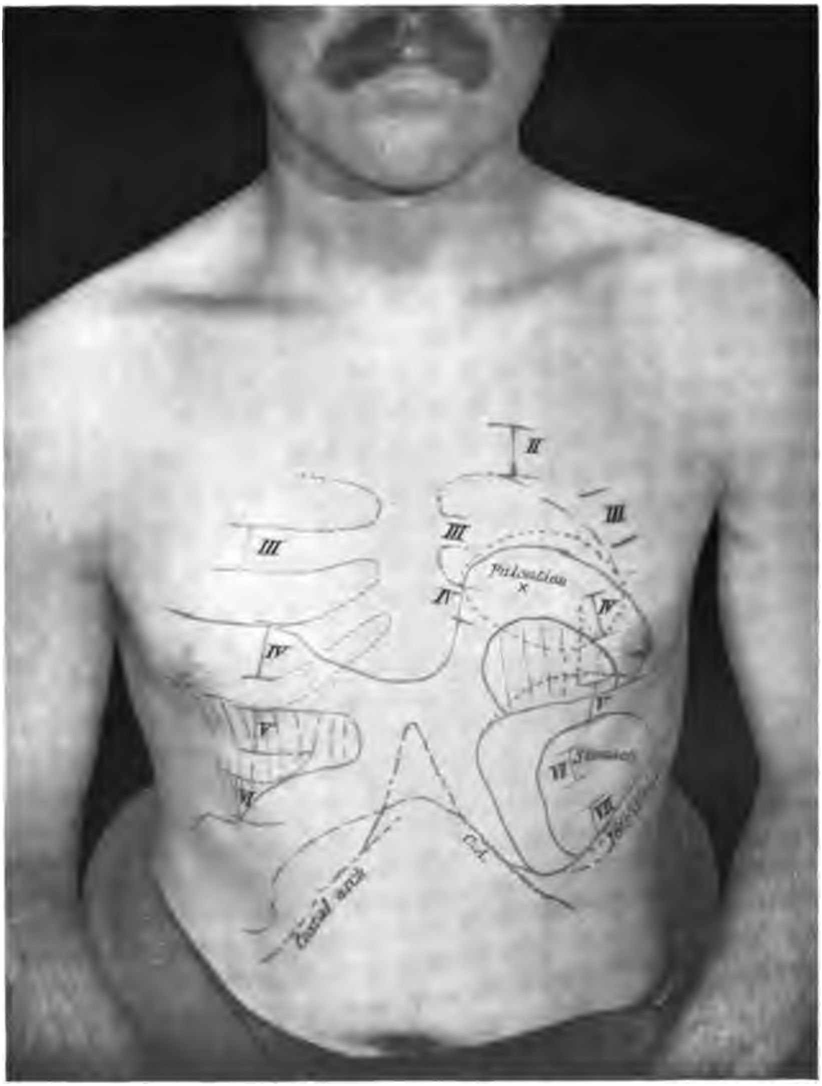

$5^{rd} = 1\frac{1}{2}'' \to 3\frac{1}{2}''$
$4^{th} = 1\frac{1}{4}'' \to 3\frac{3}{4}''$
Stomach
Intestinal resonance
1"
2
3
3
3
6
7
8
9
10

FIG. 127.

Thos. S., aged thirty. Showing high diaphragm on both sides, a small heart dullness, and a lessening of the liver dullness.

The condition of the heart on admission is shown by the outlines in fig. 127, and also by Plate II, showing a photograph of the patient after the area of heart, liver, and stomach had

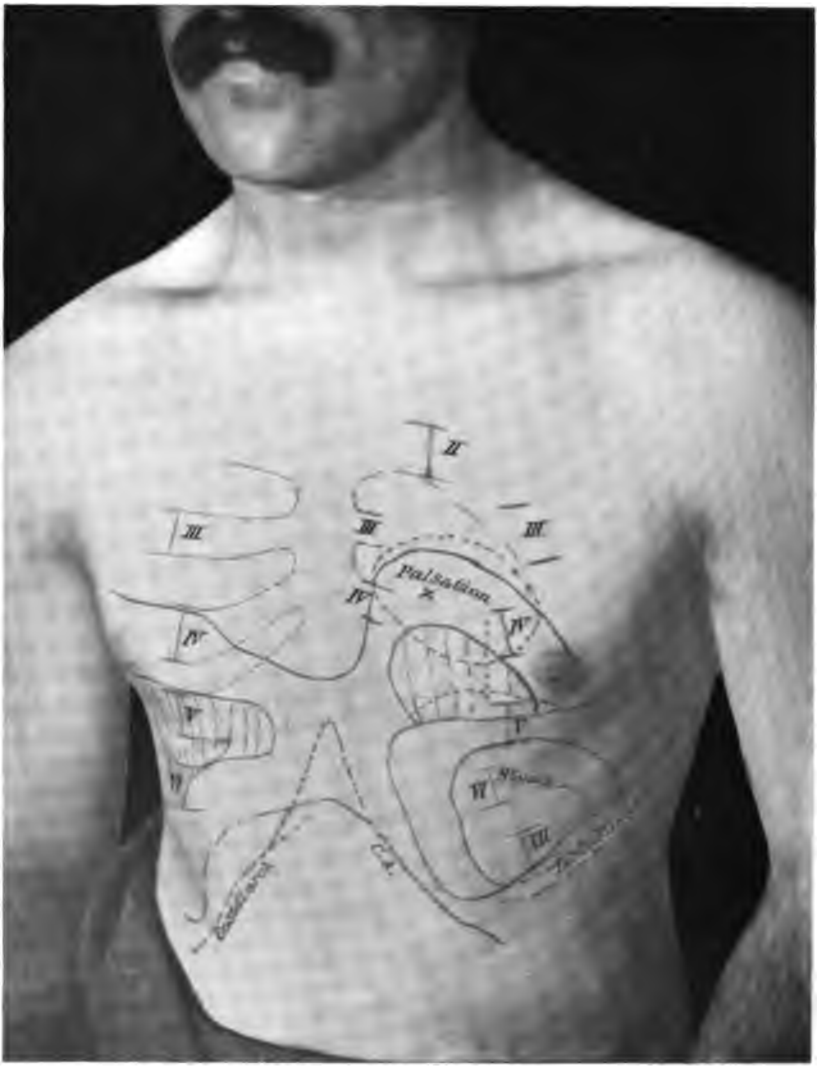
PLATE II



Photograph of the Patient Thos. S., showing the actual Outline of the Heart, Lungs, and Stomach (as determinable by Percussion) marked upon the Chest, and also the Situation of the Ribs (marked in roman numerals) and of the Costal Arch (C.A.).

Note the close correspondence of the original with the diagram shown in fig. 127; compare also Plate IV with fig. 130, p. 364. These photographs show the excellence of the diagram of the chest used in this book. It is printed by Messrs. Danielsson from a drawing by Prof. Luschka, of Vienna. It is a marvellously perfect generalisation, and in the course of nearly thirty years' experience with it the writer has very rarely indeed found it to be at fault. It has proved invaluable in the clinical studies upon which much of this book is based.

PLATE III



PHOTOGRAPH OF THOS. S., SHOWING THE OUTLINE OF THE VISCERA, AS MARKED ON THE CHEST, AS THE RESULT OF PERCUSSION.

This plate shows the extension of the gastric resonance into the axilla where it reached nearly to the mid-axillary line in the sixth interspace.

been marked on the skin. These show the diaphragm to be high and the liver dullnesss to be small. Moreover, the smallness of the stomach, combined with the high diaphragm, cause rather a remarkable shifting of the gastric resonance towards the axilla. This is well shown in the photograph (Plate III). Note the height of the relative liver dullness on the right side, the broadness of the area between the absolute and relative dullness, showing abnormal arching of the diaphragm. Note also the distance that the lower border of the liver dullness is from the costal arch, suggesting not only a drawing up of the organ by the abnormal rise of the diaphragm, but also probably a true diminution in its size, from its being underfilled with blood in common with the thoracic viscera. Also note that the amount of cardiac dullness in the third interspace is much increased (it extends to beyond the nipple line). This might be simply explained by the rise of the diaphragm lifting the heart as a whole, were it not for the fact that there is an abnormal amount of pulsation in this interspace, which readily increases in amount during any excitement, and thus shows that some upward dilatation of the right ventricle is present.

The pathology of such a case as this is not very clear. The ordinary explanation would be damage to certain of the muscular fibres of the heart as a result of the overstrain; but against this, as the prime factor, is the slightness of the actual amount of extra work that the heart did, if it is to be judged by the force put out in the single jump for life. There is, however, in the case another less evident cause of actual physical overstrain—namely, the one that is so apt to cause the death of persons with weak hearts after running to catch a train. Anyone who has tried the experiment of sitting absolutely still after the sudden cessation of violent exertion—such as running fast up a hill or upstairs—will find how greatly such an action exaggerates and prolongs the slight dyspnœa, which the exercise would otherwise have caused. The physiological reason for this is that during exercise the heart is pumping blood with full force into the muscular arterioles, which are fully dilated in response to the functional activity of the muscles. With the sudden cessation of functional activity there is sudden vasoconstriction of the muscular arterioles, and the heart which is fully distended with blood and working at high pressure will be very greatly embarrassed by the sudden closing up of the channels into which it was about to pump the blood. The sudden cessation of exertion in the case of this man as he sat absolutely still in the dark may have thrown some extra strain upon the heart, but even so it is not easy to see how such a lasting damage could be done to its muscular fibres, and there is also another point which suggests that physical overstrain was not the only or perhaps even the main factor in this case. Had muscular damage resulted from the accident the symptoms of muscular weakness surely ought to have developed at once, instead of gradually, as was the case. If, however, tachycardia played a considerable part in the case, the overstrained heart muscle would gradually get worse—as was the case here—owing to the man attempting to go on with his work, in spite of the rapidity of the cardiac action.

The case is an interesting one, and the further light which might be thrown upon it by other cases of a similar nature would be very desirable. In connection with this case it is of interest to compare the case recorded at p. 115, which appears also to have been one of ' habit irregularity ' of the heart.

ESSAY VII.—UPON DILATATION OF THE RIGHT VENTRICLE[1]

THE early recognition of slight degrees of dilatation of the right heart is of such great clinical importance that I need say nothing by way of introduction to the subject of which I am about to speak in this essay. I propose to take up in detail the special clinical features of dilatation of the right ventricle ; but before doing so it is necessary to say a word or two as to the nature and extent of this dilatation as seen in the post-mortem room.

Briefly, post-mortem study, both of the flaccid and distended organ in cases of failure of the right side of the heart, shows that the main change is an increase of the right ventricle upwards and to the left. This increase is largely due to a distension of the conus arteriosus and of the part of the anterior wall which is adjacent to it. This upward increase may be so great that the pulmonary valves lie under the first left interspace or even under the first rib, while to the left, the ventricle may extend nearly or quite to the nipple line in the second left interspace. Increase to the right of the sternum plays, as a rule, only a secondary part as compared with that in an upward direction. This statement holds true for cases due to valvular disease as well as for those where there is none.

Together with this change there is a displacement of the whole heart upwards and some rotation of the organ, so that the left ventricle assumes a position rather more posterior than normal, while the apex is tilted upwards and outwards, and is often brought into the fourth interspace in, or external to, the left nipple line. These latter changes are most evident in cases where valvular disease is not present.

---

[1] Two clinical lectures delivered at the Birmingham General Hospital, June 1894 ; printed in the *Lancet*, September 1894, and *Birmingham Medical Review*, September 1894, and reprinted here verbatim with a few omissions.

[This essay, which was written twenty years ago, gives a useful summary of much that is said at greater length in Essay I and Essay V.]

The anatomical considerations which determine the direction in which the right ventricle enlarges, as well as those which determine the displacement of the apex beat, have already been discussed (see pp. 247–254; where also the valuable pioneer work done by the late Dr. Arthur Foxwell upon this subject is referred to).

THE PHYSICAL SIGNS OF DILATATION OF THE RIGHT VENTRICLE are, to some extent, disguised or altered in cases of valvular disease by the changes in size which the left ventricle undergoes, and therefore, for the sake of clearness, I shall describe the physical signs of dilatation occurring in cases with no valvular disease—such as those commonly occurring in anæmia, chlorosis, and overstrain during adolescence, or those occurring during convalescence from rheumatic fever or the acute stage of pneumonia.

Among these cases of so-called functional dilatation a further distinction must be made, because the character varies somewhat according to the period of life and the circumstances under which it occurs—for two slightly different types of dilatation are recognisable. These may be called the adolescent and the adult type respectively.

ADOLESCENT AND ADULT TYPES OF DILATATION.—In the earlier periods of life, and especially during puberty and adolescence, the heart is naturally less resistant and less rigid than it is later in life, and is therefore more prone to excessive degrees of dilatation.

In the adolescent type of dilatation, to which my descriptions will in the main refer, the increase in size is chiefly, and often exclusively, upwards and to the left, giving an increased area of relative cardiac dullness in the third and second left interspaces and sometimes even the first interspace.

In the adult type, on the other hand, some increase of the cardiac dullness to the right of the sternum usually accompanies the upward increase, and may exceed it in amount. I need hardly point out that such divisions as these are not meant to be mutually exclusive, nor are they meant to do more than assist in the mental classification of clinical observations. Neither do I mean to imply that the adult type of dilatation is a type which occurs invariably above the age of twenty-five or twenty-six when the right ventricle fails; for in the degenerative period of life the heart may be so rigid that the patient may die with failure of the right heart, but with scarcely any clinically recognisable dilatation of the right ventricle; or, on the other hand, a man of thirty or thirty-five may have his heart substance so softened by rheumatic fever that his right ventricle may dilate just as rapidly and extensively as in any adolescent.

The physical signs of the adolescent type of so-called functional dilatation of the right ventricle are fairly definite and characteristic.

Inspection and Palpation of a well-marked case will show cardiac pulsation in the second and third left interspaces as well as in the ordinary situation; and this pulsation will be noticed to extend outwards for some 1½ or 2 inches from the sternum in the former, and to the nipple line in the latter interspace.

Further, the pulsation is not powerful like the apex beat nor like that of an aneurysm; and, as was pointed out by Dr. Foxwell (*loc. cit.*), has a very characteristic wavy appearance. The wave of pulsation may be noted sweeping downwards and outwards from the sternal end of the second left interspace. This pulsation is often further distinguishable at a glance from that of aneurysm by the fact that when the patient is recumbent the pulsation varies with respiration, being most marked with the cardiac beats which occur during the pause between inspiration and expiration, as well as with the first one to occur during the latter act. That this variation is not due simply to the position of the lungs is shown by the fact that if the breath be held at the end of expiration, the pulsation is usually less evident than during active breathing.

The pulsation can be recognised as being due to the right ventricle, and not the pulmonary artery, by the fact that it completely subsides before the occurrence of the second sound. The cardiograph also shows it to be ventricular. *Detection of the pulsation, above described, in the second left interspace* *by palpation, together with the displacement of the apex beat upwards and outwards, is often all the physical examination necessary for the diagnosis of a dilated right ventricle.*

In addition to this cardiac pulsation there will also usually be noticed pulsation in the veins of the neck—usually of the type called 'false pulsation,' and occurring only during inspiration, and due not to failure of the valves, but simply to the more rapid emptying of the distended veins which results from the aid given by the inspiratory suction to the forces which fill the right ventricle during the cardiac diastole.

As a result of careful cardiographic observation, I have every reason to believe that some of these cases of false pulsation are also largely due to a true contraction of the veins preceding and probably also accompaning that of the auricle.

Frequently the shock of the closure of the pulmonary semilunar valves can be clearly felt in the second interspace, and in extreme cases a true systolic thrill, not due to valvular disease, is present in this interspace over the pulmonary artery, and may even be detected in the subclavian and carotid arteries on the left side and much less strongly in those on the right, just as is the case with the pulmonary systolic murmur with which the thrill is associated. Pulsation may also be noted to the right of the sternum and in the epigastrium, but is less marked as a rule in either of these situations than at the base of the heart in the cases now under consideration.

The apex beat, as before mentioned, is rotated upwards and outwards so as usually to reach a point in the fourth interspace in or external to the nipple line, but in consequence of the size of the right ventricle it is often prevented from striking the chest wall and may therefore be feebly perceptible. The most prominent point of the cardiac impulse is in such a case situated internal to the nipple line and is due to the impact of the body of the right ventricle.

PERCUSSION OF THE VENTRICLE.—Although inspection and palpation may suffice when dealing with a well-marked case, it is upon percussion, aided by auscultation, that we have to rely for the early diagnosis of dilatation of the right ventricle. Since the ventricle when slightly dilated lies at some distance from the chest wall and is overlapped by lung, we must rely upon the area of relative cardiac dullness for this diagnosis; and, as a matter of practice, better results will be got by watching the relative rather than the absolute dullness, because the latter frequently does not increase at anything like the same rate as the former when the ventricle is dilating.

In ascertaining the area of relative dullness it is well to do away with the uncertainty arising from the variation in volume of the lungs by percussing the heart while the chest is held in the position of full expiration, and of course to percuss with the pleximeter finger in line with the interspace and not across it. This is my usual practice when percussing the right ventricle; and I find that while by this means a change of note can frequently be got to the right of the sternum in a normal heart it is very unusual to get any distinct deficiency of resonance at the sternal end of the second left interspace.

It must of course be borne in mind that the descent of the ribs in forced expiration will make the position of the heart appear higher relatively to them than during ordinary respiration. This alteration is not, however, as a rule, sufficient to cause any relative cardiac dullness in the second left interspace or to make the apex beat disappear from the fifth interspace if the heart be normal.

An important feature in the percussion of these 'adolescent' cases, and one which has important bearings upon the causation of the basal systolic murmur of anæmia and debility, is the fact that the cardiac dullness in the second and third left interspaces is increased when the patient lies down. The difference is often considerable, and there frequently is as much as an inch more relative dullness in the second space when the patient is recumbent than there is when in the erect position.

Percussion of the heart in dilatation of the right ventricle shows that the area of relative dullness increases upwards into the second space and broadens outward in the third and fourth spaces, until in advanced cases the left edge of the dullness almost coincides with the left vertical nipple line from the second to the fourth spaces inclusive. At the same time there may be a slight increase of the relative dullness to the right of the sternum, but it rarely exceeds one inch; on the other hand, there often seems to be an actual diminution of dullness in this direction. The absolute cardiac dullness broadens and also extends upwards, and may in advanced cases reach a width of one inch in the second left space; it very rarely extends to the right of the sternum.

Auscultation.—The diagnosis of dilatation of the right ventricle by means of the stethoscope is not so satisfactory as by the methods just discussed, for the auscultatory phenomena are not always definite and constant.

Among the physical signs which may be looked for are the following:—

1. The well-known pulmonary systolic murmur, usually heard best in the second left interspace.

2. Accentuation of the pulmonary second sound, and occasionally true reduplication of the second sound at the base of the heart.

3. The venous hum, so well known in cases of anæmia, and called the *bruit de diable*.

4. The murmur due to tricuspid regurgitation.

5. Less frequently a false reduplication of the second sound, audible only over the ventricle and constituting one of the varieties of *bruit de galop* or 'triple sound.' This sound is quite distinct in origin, in time, and in area of audition from the second sound, and I have for several years called it the 'third sound' of the heart.

6. More rarely still a tricuspid diastolic murmur accompanying or replacing the third sound above mentioned.

1. The Pulmonary Systolic Murmur.—The most reliable and constant of the above-mentioned phenomena is the well-known systolic murmur audible at the base of the heart to the left of the sternum, and correctly designated the pulmonary systolic murmur. This murmur is of considerable diagnostic value in the dilatation which occurs as a result of overstrain with or without chlorosis or anæmia in adolescence. Its loudness may be taken as a reliable guide to the amount of the upward enlargement of the ventricle which is present. A good illustration of this is the increase in the loudness of the murmur which accompanies the increase of the cardiac dullness when the patient lies down. The murmur is also markedly increased

by exertion, and this is apt to lead to a fallacy in connection with the statement last made, for it will be increased in loudness by any sudden change of position.

The above statement that the murmur is proportional in loudness to the degree of upward increase of the dullness does not always hold true for acute cases occurring in pneumonia and allied conditions, for in them the murmur may be faint although the amount of dilatation be considerable.

Speaking, however, of the before-mentioned non-febrile cases of dilatation in adolescents we can say that the pulmonary systolic murmur is a good indication of the presence and amount of dilatation in any particular case, and gives the first sign of commencing dilatation and is the last sign to disappear on its subsidence. In slight cases it is only audible when the patient is recumbent, especially after considerable exertion, and is then soft and faint, and its area of audition is confined to a small area one inch or so in diameter at the sternal end of the second left interspace. When well marked, the murmur is blowing in character, resembling the ordinary mitral systolic murmur, except that the pitch of the former is often lower, and may sometimes be almost humming in character. Its point of maximal intensity is over the second left interspace, usually at its sternal end, although when the dilatation is considerable this point may be as much as one inch or more from the sternum.

DIRECTION OF CONDUCTION.—It is conducted upwards and to the left to a greater extent than downwards and to the right: in fact, unless very loud it is usually not heard below the third interspace. When loud it is audible into the subclavian and carotid arteries, although it is better conducted to those of the left than those of the right side. When very loud it is even audible into the axillary arteries and over a great portion of the upper part of the chest anteriorly. It may even be accompanied by a distinct systolic thrill, which in extreme cases may be palpable over a wide area and follows the same lines of conduction as the murmur.

ETIOLOGY OF THE PULMONARY SYSTOLIC MURMUR.—The exact cause of this murmur is not perfectly certain, but the pathological condition on which it depends seems, as was pointed out by Dr. Foxwell (*loc. cit.*), to be the upward displacement of the anterior portion of the root of the pulmonary artery. In consequence of the upward dilatation and displacement of the anterior wall of the right ventricle, while the interventricular septum which forms the posterior wall remains fairly normal, there is some distortion of the pulmonary artery. This seems to be of two kinds: Firstly, alteration in the direction of the artery in relation to the line of the ventricular cavity, in consequence of the artery having to reach its fixed point (namely, the bifurcation of the trachea) from a point under the first interspace, instead of running its usual course from under the second interspace to the bifurcation. Secondly, a potential redundancy of tissue in its anterior wall. Normally, the anterior wall runs from a point subjacent to the middle of the third costal cartilage in a curved course to the point where the artery bifurcates close to the spine—a distance, say, of two inches and a half. When, however, the right ventricle is dilated the anterior wall of the artery commences at a spot nearly an inch nearer to the point of bifurcation than normal, while the posterior wall retains more nearly its normal relations. The result of this is that when the artery is full the anterior wall, being somewhat relaxed, will distend more easily than is normally the case, and therefore during each systole there will be formed an aneurysm-like dilatation of the artery. We would expect this bulging to be more marked in the recumbent than in the erect posture, because in the latter case gravitation tends to prevent the heart from taking quite such a high position as it would otherwise do. Strong evidence in favour of the above theory is given by the fact that the murmur increases in loudness proportionally to the increase in dullness which occurs when the patient lies down, the murmur always being louder when the patient is recumbent than when erect.

According to this theory, the conduction to the vessels of the neck is of course due, as was pointed out in the paper above referred to, to the contiguity of the aorta and pulmonary artery. If, post-mortem, both sides of the heart be distended with hard paraffin before the removal of the heart and lungs from the body, the anatomical reason for this conduction at once becomes apparent, as well as that for the better conduction of the murmur to the arteries of the left side than to those of the right. The production of a thrill is easy of explanation by this hypothesis, for there is an aneurysmal dilatation of the pulmonary artery into which the blood enters in a direction nearly at right angles to the line of the artery.

2. ACCENTUATION OF THE PULMONARY SECOND SOUND.—Accentuation of the pulmonary second sound is of course of extremely common occurrence in these cases, but is of little *diagnostic* value so far as the presence or absence of dilatation is concerned. The point of maximal intensity of the second sound is however of value as giving an indication of the situation of the pulmonary valves. Thus in dilatation of the right ventricle the P.M.I. of the second sound is usually higher than normal, and as the dilatation subsides this point may be observed to alter its position and descend towards its normal situation.

The degree of accentuation of this sound gives us also important information as to the tension of the blood in the pulmonary artery, and shows to some extent the amount of work which the ventricle is doing.

3. BRUIT DE DIABLE.—The venous hum usually associated with anæmia, which is audible at the root of the neck over the great veins and is commonly called the *bruit de diable*, is also intimately related to failure of the right ventricle, although I do not think there are sufficient reasons for thinking that it cannot occur apart from this failure.

As is well known this murmur is best heard in the erect position, and disappears to a greater or less extent—often entirely—when the patient lies down. The pathological explanation of this is at once made evident if at a post-mortem on one of these cases the trouble be taken to distend the right heart and great veins with hard paraffin before its removal from the body. Such an injection shows very beautifully the constriction of each dilated vein at the point where it joins another vein, due to the mechanical support afforded at these spots. A murmur must arise on the proximal side of each of these constrictions as the blood flows towards the heart.[1] When the patient is erect the action of gravitation is to increase the pressure of the blood flowing into the innominates and superior cava from the head and neck, thus increasing the distension of the veins. When the patient is recumbent this pressure is lessened, and the veins near the heart being less distended, the relative constriction at the points of junction is less and the venous hum lessens or disappears as a consequence.

The clinical importance of the venous hum is that it shows the existence of want of tone in the great veins. This fact may be of considerable importance if, as seems to me probable, the contraction of the veins plays an important part not only in the filling of the auricle, but also in checking tricuspid regurgitation by means of a more prolonged and powerful contraction than normal.

4. THE MURMURS DUE TO TRICUSPID REGURGITATION.—Theoretical as well as practical considerations seem to show that tricuspid regurgitation must accompany dilatation of the right ventricle (of the adult type)—at all events, if it be well marked. But from a clinical point of view it is sometimes difficult to point to any physical sign which makes its presence or absence certain in any particular case. It can, I think, be truthfully affirmed that there is no physical sign which is characteristic of tricuspid regurgitation in the way in which the systolic apical murmur with displacement of the apex beat is characteristic of mitral regurgitation.

Perhaps the two most reliable signs are—firstly, the presence of a systolic murmur audible in any or all of the following situations: (*a*) over the body of the right ventricle, or (*b*) over the right auricle, and also (*c*) over the situation of the superior vena cava just to the right of the sternum in the first and second interspaces; and secondly, the presence of true systolic pulsation in the veins of the neck or over the liver.

The diagnostic value of the former sign is diminished by the fact that it may sometimes be absent in even well-marked cases of tricuspid regurgitation (*vide* p. 280).

True pulsation in the veins of the neck is almost absolutely reliable as a positive sign of tricuspid regurgitation, but its absence is valueless as a negative sign. Very frequently cases of undoubted tricuspid regurgitation are met with in which this sign is absent. As a most marked instance, I may quote the case of a girl aged fifteen, who was under my care in this hospital, suffering from mitral disease, with an enormously dilated heart. The apex was in the axilla 6½ inches from the sternum. The cardiac pulsation was visible from the second to the seventh interspaces inclusive, and extended to the right of the sternum as far as the right nipple line (2½ inches). The pulsation to the right of the sternum was shown by the cardiograph to be due to the auricle, and there was so much tricuspid regurgitation that a systolic thrill was clearly felt over the auricle in the neighbourhood of the right nipple; nevertheless, there was no venous pulsation in the neck, nor was there any pulsation of the liver during the six months that she was under observation (*vide* p. 404, Case V, and Plate XII).

---

[1] The fact that the murmur is slightly louder during diastole than during systole is in harmony with this theory of its production.

SYSTOLIC TRICUSPID MURMUR.—In cases where there is no true pulsation in the veins of the neck the tricuspid systolic murmur is often of great diagnostic value. The chief point of difficulty is its distinction from the pulmonary systolic murmur. The main distinguishing features are as follow: The area over which it is heard is, as a rule, lower than that of the pulmonary murmur. The latter is rarely heard below the third space, whereas the area of audition of a pure tricuspid murmur is approximately an oval-shaped area bounded on the right by the sternum, above by the third rib, below by the fifth rib, and extending a variable degree towards the nipple line in the fourth space. This area varies according to the amount of dilatation present, and may be approximately put down as coinciding with the inner half or two-thirds of the area of the absolute dullness.

A difference in pitch between the two murmurs will sometimes be noted by which they can be distinguished, but the character of the tricuspid murmur varies much in different cases.

A point of diagnostic value is sometimes obtained in the fact that a tricuspid regurgitant murmur is heard very loudly at the sternal ends of the second and third right interspaces over the situation which the tip of the right auricular appendix would occupy in a dilated heart. In my experience it is, however, unusual to find the murmur audible here in the adolescent type of functional cases. It is more common in valvular disease and with the adult type of dilatation.

When the murmur is audible in both the first interspaces over the great veins it is very difficult to distinguish it from an aortic murmur. The point to be relied upon is its loudness close to the sternum as compared with its feebleness or absence over the carotids and distal portions of the subclavians; a moderately loud aortic systolic murmur being of course loudly conducted along the arteries.

5. THE THIRD SOUND OF THE HEART.—By this name I consider that we ought to designate the sound which when added to the second sound of the heart gives rise to what is known as 'a reduplication of the second sound which is audible only at the apex.' The occurrence of this sound at the apex or over the right ventricle of the heart produces one of the varieties of what is known as the *bruit de galop*, or triple sound of the heart. The most familiar instance of its occurrence is in mitral stenosis, when there is audible at the apex a presystolic murmur and 'reduplicated second sound.'

It is of course an abnormal sound in the human heart. As regards the time of its occurrence, it follows the second sound, the interval being nearly equal to that between the first and second sounds, and, moreover, in any one case the interval appears to be constant and does not vary when the heart is irregular.

In character it is a faint sound, and is often, in the cases we are dealing with, inaudible with a wooden stethoscope or when the binaural stethoscope is pressed too firmly. It is very apt to be inconstant in its occurrence, and frequently is only to be heard with the heart beats which occur during the pause between inspiration and expiration and at the commencement of expiration. Its area of audition is over the body of the right ventricle, and it is not loud enough to be conducted widely.

In the cases we are now considering, clinical observation seems to show that the two factors upon which this sound depends for its production are the amount of dilatation of the right ventricle and the strength of the forces producing the inrush of blood into the ventricle on the opening of the tricuspid valve. Thus in a feebly acting and dilated ventricle, we get no third sound, but as the strength of the heart increases, a third sound often appears for a time and disappears again when the ventricle further regains its tone and the dilatation disappears.

I am unable here to go fully into the causation of this 'third sound,' and I will only say that I feel sure that this sound, as well as the diastolic murmur, which is often closely associated with it and which will shortly be mentioned, are both of them manifestations of an active expansion movement on the part of the ventricle whereby blood is sucked into it from the auricle and veins immediately after the closure of the semilunar valves (*vide* p. 427).

The most marked instance of this third sound and murmur occurs in the left ventricle in mitral stenosis, for there the valves are permanently prevented from assuming their normal position as the ventricle enlarges during its expansion movement, and the ventricle is, moreover, not only of normal strength, but is able also to alter the mode and strength of its contraction so as to develop a greater amount of suction, as is evidenced by the increased loudness and sharpness of the first sound in mitral stenosis.

6. The Diastolic Tricuspid Murmur.—This murmur is produced in the same manner as with the third sound of the heart, with which it is so closely associated clinically. It only remains to say that the murmur is usually faint and low-toned or humming, and is audible simply over the body of the right ventricle. The outer limit of its area of audition is often so sharp as to suggest the probability that when present the localisation of this murmur may serve as a guide to the position of the interventricular septum. As regards its occurrence, it is rare in functional dilatation in adolescents, and I have only observed it three or four times out of several hundred cases carefully examined. In dilatation of the right ventricle, secondary to valvular disease, it is much more frequent.

The Adult Type of Dilatation.—I think I have said enough already as to the points which separate the adolescent type, which I have been discussing, from the adult type, to make further dwelling upon this subject unnecessary.

DILATATION SECONDARY TO VALVULAR DISEASE.—Most of what I have already said will be found to apply to this form of dilatation if allowance be made for the effect of the changes in the left side of the heart which result from the primary lesion. The main difference beween this and the former class of case is that the ventricle here is not necessarily weaker than normal, and is of course often hypertrophied. Speaking broadly, the dilatation of the heart to the right of the sternum is more apt in these cases to equal or exceed in amount that in the second and third left interspaces; and further, the systolic tricuspid murmur is in these cases more marked, and the pulmonary systolic murmur less likely to be present than in the functional cases. Other points of distinction may be gathered from what I have already said.

SYMPTOMS. — The symptoms of dilatation of the right ventricle are mostly of the usual type—namely, breathlessness, palpitation, &c. There is one symptom, however, which is fairly distinctive of the adolescent type, and that is—pain localised to the region of the second and third left costal cartilages. This pain may be fairly acute, but more usually it consists of a dull pain, and is frequently accompanied by a sense of tightness in this region; and there is often in the adolescent class of cases a feeling as if too large a bolus of food had been swallowed and was sticking in the throat at the level of the second left interspace. These sensations are often relieved somewhat when the patient takes a prolonged deep inspiration, and this sighing voluntary inspiration may often be noted in these cases. In addition, there is sometimes tenderness on percussion over the dilated ventricle in the second left interspace, and there may even be tenderness on pressure.

ETIOLOGY OF DILATATION OF THE RIGHT VENTRICLE.—Failure of the right ventricle will result from (1) excessive work thrown upon the normal ventricle—i.e. absolute overstrain; or (2) normal amount of work thrown upon a weakened ventricle—i.e. relative overstrain.

1. ABSOLUTE OVERSTRAIN.—In this class of case the

cause of the failure and dilatation of the ventricle is for the most part not inherent in the ventricle itself. The most typical examples of this class are those in which there is dilatation of the right ventricle as a result of overstrain from excess of athletic exercise or from excess of heavy work—such as lifting heavy weights—in otherwise strong and healthy young men. These cases may be subdivided into—(*a*) those in which the strain upon the right ventricle is indirect and due to the demands of the systemic circulation, as is the cases above mentioned, or in valvular disease of the heart; or (*b*) those in which the strain is due to primary embarrassment of the pulmonary circulation. Of this the most typical instance is of course emphysema. Pneumonia also produces dilatation from this cause, and I believe that we must also include in this category many, if not most, of the common cases of dilatation of the right ventricle in anæmia and chlorosis. Of course, in these last-named cases, a prominent feature is the weakness from the anæmic state of the blood; but I think we have sufficient ground for believing that there is in addition an abnormal amount of contraction of the pulmonary arterioles (or venules) caused by the poor state of the blood. This is a subject for physiological experiment. The fact that in suffocation there is a spasm of the pulmonary arterioles which is removed by nitrite of amyl, as has recently been pointed out by Sir George Johnson, is in favour of the above supposition. Two years ago, in my thesis for the degree of M.D., I expressed my belief that the pulmonary vasomotor system had in all probability much to do with the failure of the right side in anæmia; but unfortunately ill-health has prevented me from investigating this subject further, except so far as to enable me to say that there seems every probability that nitrites may prove of considerable value in these cases.

2. Relative Overstrain as a Cause of Dilatation of the Right Ventricle.—As a typical instance of this, I may give the dilatation which is apt to occur after the fall of the temperature in rheumatic fever, but more especially when the patient first begins to get up. In such a case, I have seen the right ventricle dilate out nearly to the left nipple line in the second left interspace in the course of a day or two.

The dilatation in cases of anæmia and chlorosis is also in part due to weakening of the ventricle from malnutrition; and, as another instance, may be mentioned the weakening due to many acute febrile conditions.

In conclusion, I may say that we have much yet to learn as to these changes in the right side of the heart, and that there is scope for much careful clinical study as to the effect of drugs upon the pulmonary circulation, upon the great veins, and upon the character and strength of the important expansion movement of the ventricle.

Essay VIII.—ON DISPLACEMENT OF THE HEART

This essay will be chiefly devoted to those kinds of displacement which are of both interest and importance to the clinician, and it is not intended to take up cases where the interest in mainly anatomical, such as the changes which take place in the situation of the heart and the relative position of its chambers in spinal curvature. Moreover, as this essay is based entirely upon observations by the bedside, the question as to the amount and the degree of rotation of the heart upon itself which results from lateral displacement of the organ is scarcely touched upon.

Clinically, all cases of cardiac displacement may be divided into two main groups:—

Firstly, those where the position of the heart in the thorax is altered as a result of a change in the heart itself or its great vessels (e.g. by aneurysm). In this case the cause of displacement may be said to be intrinsic.

Secondly, where some extrinsic cause of displacement is at work and the position of the heart is altered as a result of changes in the surrounding viscera (e.g. displacement of the heart by a pleural effusion).

I. Cardiac Displacement due to Changes within the Heart Itself

Amongst the intrinsic causes of displacement of the heart, a common one is that due to aneurysm. As regards the displacement due to aneurysm of the aorta arising outside the pericardium no serviceable generalisation can be made, for the amount and type of displacement depends upon the size of the aneurysm and the situation where it develops.

In the case of intra-pericardial aneurysm arising in or near the anterior sinus of Valsalva, a characteristic displacement seems to result—although the writer's experience has been too limited to make a certain generalisation.

An aneurysm arising in this situation seems to displace the right auricle downwards and to the right, and the right ventricle outwards to the left and downwards. Such a displacement is shown in the accompanying figure—drawn from a photograph taken at the post-mortem examination of a case of intra-pericardial aneurysm (*vide* fig. 128).

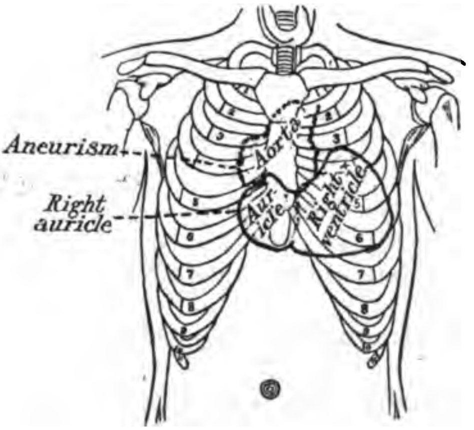


Fig. 128.

Situation of heart showing downward displacement due to intra-pericardial aneurysm. (Made from a photograph.)

Closely allied to the displacement of the heart due to aneurysm is that noticeable in cases of aortic regurgitation.

In many text-books the fact is noted that the apex of the heart in well-marked aortic regurgitation lies at a relatively lower level than is the case when the enlargement is due to mitral regurgitation, and it is stated that whereas in the latter the apex of the heart lies in or external to the original axis of the heart, in aortic regurgitation, on the other hand, the apex is apt to be internal to the original axis—i.e. lies at a relatively lower level than in mitral regurgitation. Various reasons for this are given, but I have not yet come across the following, which seems the true explanation.

In such cases as these it can, as a rule, be easily recognised that the point of maximal loudness of the pulmonary second sound, as well as the upper border of the cardiac dullness, are both lower than normal, and therefore it is justifiable to state that the lowering of the apex beat is in all probability due to a displacement downwards of the whole heart rather than simply to a special downward increase of the left ventricle alone.

The explanation of this downward displacement is to be found in the fact that over-distension of the first part of the aorta is a necessary consequence of the establishment of compensation in well-marked aortic regurgitation.

The reason for this may be given shortly as follows :—

It may be taken for granted that, when the muscular power of the heart is good, a definite amount of blood (approximately 3 ounces) ought to pass into the arteries at each contraction of the ventricle.

In order to bring this about, when there is leakage of some of the blood back through the aortic valves at each beat, it is necessary for the ventricle to throw forward with each contraction an extra amount of blood in order to allow for the leakage. This extra amount must be at least equal to the normal amount plus the amount of the leakage. Therefore, if at each beat half an ounce leak back, the ventricle must throw rather more than 3½ ounces into the aorta at each contraction if 'compensation' is to be established. In other words, the process of compensation in aortic regurgitation demands that an amount of blood considerably above the normal must be thrown into the aorta at each beat, and it must therefore be abnormally distended by the ventricular systole.

Now, as fluid pressure acts equally in all directions, this distensile force will not only tend to produce the well-known increase in the transverse diameter of the aorta, but also to stretch it longitudinally as well, and the lengthening of the aorta which must be thus brought about must tend to displace the heart downwards. I have no doubt that careful measurements of the level of the aortic valves post-mortem in a series of cases of aortic regurgitation would prove definitely that this is so.

Owing, however, to the elasticity of the aorta it is not likely that this downward displacement of the heart would be as evident after death—when the distensible force of the blood pressure is no longer acting—as it is during life.

Of all the intrinsic causes of alteration in the position of the heart, by far the commonest is the upward displacement of the apex which occurs in anæmia.

The late Dr. Foxwell was, I believe, the first to point out the frequency of this phenomenon and its probable cause (*vide* his 'Essays in Heart and Lung Disease,' p. 343).

The subject of upward displacement is discussed at length in the following pages.

UPWARD DISPLACEMENT OF THE HEART BY INTRINSIC CAUSES

The intrinsic causes of elevation of the apex are of considerable interest and importance clinically. In the first place, dilatation of the right ventricle downwards may, under certain circumstances, cause a recognisable degree of elevation of the apex. Since the right ventricle occupies the inferior aspect of the heart and rests upon the diaphragm, any abnormal fullness of this chamber must, theoretically, raise the apex of the heart to some extent; but this rise, due to right ventricular dilatation, would not be recognisable clinically, being so small. When, however, the heart's fixation to the diaphragm by the inferior vena cava is taken into account it will be seen that, taking this point as the fulcrum of a lever, the effect of a bulging of the right ventricle at a spot near the fulcrum will cause a much greater effect at the cardiac apex, which lies at a considerable distance from the fulcrum. If the point where the dilated right ventricle presses on the diaphragm lies, say, 2 inches away from the fixation point (inferior vena cava), and the apex lies 6 inches away, any downward dilatation of the ventricle would give a threefold effect at its apex, and an increase of ¼ inch in the right ventricle (supposing the diaphragm to be unyielding) would give a ¾ inch rise of the apex.

As the structures concerned are not rigid, but elastic, it is not easy to say how far the mechanical principles just enunciated will apply; but occasionally a case is met with where the upward displacement of the apex appears to be due to the cause here suggested.

Another intrinsic cause of elevation of the apex is found in the changes which take place in the heart when it fails and dilates as a result of anæmia in adolescence, and this cause of elevation of the apex is of considerable clinical interest and importance.

Although the fact of the elevation of the apex is clearly demonstrated by clinical observation, the explanation of the mechanism whereby it is brought about is neither clear nor easy of explanation, although it certainly seems due to changes within the heart itself.

The clinical facts upon which any theory must be based are these :—

1. In anæmic dilatation of the right ventricle—such as is seen in adolescents—the apex is frequently raised above the normal level. The careful study of 192 patients with well-marked dilatation showed that the apex was raised above the level of the fifth interspace in 87 out of 192 observations, and normal in 105—i.e. it was distinctly raised above the normal level in 45·3 per cent. of the cases.

2. As regards age as a factor in this rise. In the first place, Is the apex more displaced in more distensible or in less distensible hearts? To try to answer this question, I have tested the relationship which the age of the patient bears to the presence or absence of upward displacement of the apex. It was more frequent in patients between twenty and twenty-five years of age than in younger patients; for 58 per cent. of the former and less than 40 per cent. of the latter had a raised cardiac apex.

The figures available are as follows :—

Age	No. of cases	With raised apex	With normal apex
13 to 19	103	41 (= 39·8 %)	62 (= 60·2 %)
20 to 25	75	44 (= 58·6 %)	31 (= 41·4 %)
	178	85	93

The following are more detailed figures :—

Age		No. of cases	With raised apex	With normal apex
Younger group	13 to 15	12	6	6
	16 to 18	59	22 (= 37 %)	37 (= 63 %)
	19	32	13 (= 40 %)	19 (= 60 %)
		103	41 (= 39·8 %)	62 (= 60·2 %)
Older group	20 to 21	42	27 (= 64·2 %)	15 (= 35·8 %)
	22 to 25	33	17 (= 51·5 %)	16 (= 48·5 %)
		75	44 (= 58·6 %)	31 (= 41·4 %)

Again, the fact that a raised apex-beat is more likely to be found in an older than a younger adolescent with anæmia is shown by an analysis of the whole of the 85 cases with a raised apex-beat. Fifty-seven of them, or 67 per cent., were nineteen years of age or more, and only 33 per cent. were under nineteen, whereas in the group with normal apex 54 per cent. only were in the older group and 46 in the younger.

But age as an etiological factor ought also to be studied in relation to the amount of the right ventricular dilatation present in the cases. For this purpose, I here give the average amount of relative cardiac dullness which was detected in the second left interspace when the breath was held in expiration.

In this connection, I have eliminated those cases where there was no marked dilatation of the heart upwards, because in none of these was the apex above the normal level.

In this way the available cases are reduced to 114.

I have divided them into four groups—namely, those fourteen to fifteen years of age, those sixteen to eighteen, nineteen to twenty-one, and twenty-two to twenty-five respectively; and the subjoined table shows the number of cases in each group in which the apex was raised above the fifth interspace and also the average amount of relative cardiac dullness in the second left interspace in each group.

Age	No. of cases	No. with apex in fourth space	No. with apex in fifth space	Average amount of dilatation in second space
14 to 15	7	3	4	1·2 inch.
16 to 18	40	11	29	1·07 „
19 to 21	48	27	21	1·37 „
22 to 25	19	11	8	1·1 „
	114	52	62	Average dilatation 1·2 inch

These results seem to show that in patients over twenty-one years of age there is a greater tendency for the apex to be displaced upwards when there is only a moderate degree of upward dilatation than there is in the younger patients.

In the case of those aged nineteen, twenty, and twenty-one, a reliable comparison is not possible, owing to the greater amount of upward dilatation which occurred in them.

The question of the bearing of the age of the patient upon the displacement of the apex may be studied in another way —namely, by dividing up the cases into groups according to the amount of upward dilatation present, and then noting the relative ages of the patients, showing upward displacement of the apex, as compared with those whose apex beat was in the normal situation.

The results are as follows :—

Amount of dilatation	Total number of cases	Average age of group	No. of cases with apex in fourth space	Per cent.	Average age	Cases with normal apex	Average age
1 inch or less	67	19·4 yrs.	26	38·8	19·8 yrs.	41	19·3 yrs.
Over 1 inch	55	18·9 yrs.	32	58·1	19·3 yrs.	23	18·1 yrs.
Dilatation in third space only	24	20·6 yrs.	8	33·3	22·5 yrs.	16	19·7 yrs.
	146		66			80	

These tables show clearly that the age of the patient is an important factor in determining whether the apex will be raised or not in a case of upward dilatation of the right ventricle. Thus, in a group of 55 patients who showed more than 1 inch of relative cardiac dullness in the second left interspace and whose average age was 18·9, the average age of the 32 patients who showed a raised apex beat was 19·3 years, whereas that of the 23 patients who had a normal apex beat, was only 18·1 years.

In the group of patients with 1 inch or less, the ages of those with raised apex and normal apex are practically the same—namely, 19·8 years and 19·3 years respectively.

In the last group with which the table deals—namely, those showing no dilatation in the second left interspace, but some dilatation in the third, the greater age of the patients with a raised apex beat is very marked ; but as the group is only a small one too much stress cannot be laid on the results.

They are, however, striking—namely, the eight cases with upward displacement of the apex averaged 22·5 years of age and the sixteen with normal apex averaged only 19·7.

These facts seem to suggest that the displacement of the apex is associated with a stretching of the *whole* anterior wall of the ventricle; for this type of stretching is more likely to occur in the less distensible hearts of girls of twenty rather than in the more distensible hearts of younger girls, for in them the upper part of the anterior wall only is the most apt to dilate.

1. Amount of Upward Dilatation as a Factor in causing Upward Displacement of the Apex

The table just given shows clearly that the apex is, *ceteris paribus*, more apt to be displaced in cases showing much dilatation than in those showing little dilatation. Thus out of 67 cases, where there was 1 inch or less of dilatation, the apex was displaced up in 26—i.e. in 38·8 per cent.; whereas out of 55 cases showing more than 1 inch of relative dullness the apex was displaced up in 32 cases, or 58·1 per cent.

Therefore, there seems to be a direct relationship between the amount of dilatation in the second left interspace and the liability of the apex to be displaced upwards. It must, however, not be forgotten that this is only *one* factor, and that great upward dilatation of the right ventricle can exist without any upward displacement of the apex.

2. Broadening of the Cardiac Dullness to the Left as a Factor in causing Upward Displacement of the Apex

In certain cases, as already pointed out, the right ventricle dilates outwards to the left, causing an abnormal broadening of the cardiac dullness in the third left interspace, which may or may not be accompanied by an extension of the right ventricle upwards into the second interspace.

We have seen in the last table that where this type of dilatation occurs the apex was elevated in five out of fourteen cases namely—35·7 per cent.; but it will be interesting to see whether upward displacement of the apex is more frequent where this type of dilatation accompanies the upward distension of the ventricle than where it does not.

For the purposes of this inquiry, the available cases are reduced to forty-three, because all those in which there was more than 1 inch of relative dullness in the second left interspace have broadening of the cardiac dullness in the third interspace and are therefore useless for comparison. Other cases are not available, owing to absence of any note as to the amount of dullness in the third left interspace.

Of the 43 cases where there was 1 inch or less of relative dullness in the second interspace 26 showed dilatation in the third interspace and 20 showed a normal amount of cardiac dullness.

Of the 26 with dilatation in the third interspace the apex was displaced upwards in 14, or more than half, and normal in 12 out of the 26; whereas out of those with normal dullness in the third interspace there was upward displacement of the apex in only 7 out of 20 cases, or about one-third, showing as before that an increase of the cardiac dullness to the left is more apt to accompany elevation of the apex than is the case with upward dilatation.

Further evidence as to the causal relationship between the dilatation of the right ventricle and the displacement upwards of the apex is furnished by the fact that out of the 220 cases of heart failure in anæmia there were only two in which the apex of the heart was raised above the normal position in the absence of dilatation of the ventricle upwards and to the left.

Once, in the case of a patient aged twenty-four, with some tricuspid regurgitation, where an unrecognised downward enlargement of the right side of the heart might have lifted the apex (*vide* p. 237), and once in the case of a patient whose physical signs were anomalous in other ways, there was a well-marked systolic pulmonary murmur, but no upward or outward dilatation of the right ventricle, and the murmur was, moreover, louder in the erect than in the recumbent attitude. For the further discussion of this case, *vide* p. 274.

There is another possible factor at work in the upward displacement of the apex in these cases of anæmic heart failure, but I am not able at present to speak definitely for or against its importance. I refer to a possible diminution in the size of the left ventricle as a contributory factor towards the raising of the apex.

As has been pointed out (p. 243), there is distinct evidence of a hindrance to the passage of the blood through the lungs in many cases of anæmia, and it may be that this, coupled with the weakness of the right heart, may lead to a lessened supply of blood to the left side and a consequent diminution in its size.

Careful study of the size of the liver and the level of the diaphragm throughout a series of cases would tend to clear up this point.

3. Dilatation to the Right not a Factor in the Causation of a Raised Apex

A rise in the level of the apex of the heart is not as a rule associated with dilatation of the right ventricle to the right of the sternum, and this type of dilatation does not play any part in the causation of the apex rise. It is in fact less frequent among cases with a raised apex-beat than among those with the apex at a normal level.

The figures are as follows:—

Taking as normal, cases where there was ½ inch of relative dullness to be detected to the right of the sternum on full expiration, and as abnormal those cases which showed more, the following facts are obtained: Out of the 85 cases with a raised apex beat there were only 5 which showed an abnormal amount of cardiac dullness to the right of the sternum—that is, 5·8 per cent. Whereas among the 93 cases with the apex at a normal level there were 10 with dilatation to the right, or 10·7 per cent.

If the cases with a raised apex be further analysed it will be found that those showing dilatation are more numerous among the older cases of anæmia. In those of eighteen or under (28 in number), there was only one case of dilatation of the heart to the right; whereas among the 57 cases nineteen years of age or more there were 4 cases of dilatation, or 7 per cent.

Among the cases with the apex at a normal level 4 out of the

43 patients who were eighteen or less had some dilatation—i.e. 9·3 per cent.; and out of the 60 in the older group, 6, or 10 per cent., had some dilatation. Therefore, these figures show that dilatation to the right is less frequent when the apex is raised than when it is at a normal level, and they also show clearly the extreme infrequence of dilatation to the right in those cases of cardiac failure due to anæmia in young patients. For in the cases with a raised apex the right border of the heart was normal in 94·2 per cent. and in those with a normal apex it was normal in 89·3 per cent.

4. TRICUSPID REGURGITATION NOT ASSOCIATED WITH A RAISED APEX

As regards the presence of a tricuspid murmur in cases with a raised apex beat it can also be said that the changes in the heart which produce this murmur do not act as factors in the causation of the upward displacement of the apex.

Amongst the 178 cases, there was a systolic murmur present in 41 instances, or 23 per cent., and of these the murmur is noted as being faint in 12 instances. The cases with tricuspid murmur are fairly equally divided between those with an upwardly displaced and those with a normal apex beat—namely, 21 among the former and 20 among the latter; giving a greater frequency among those with a raised apex—i.e. 24·7 per cent. as against 21·5 per cent. If, however, only cases with a well-marked murmur be considered, the frequency of occurrence of such a murmur is the same in both classes—i.e. 16·4 per cent. in those with a raised apex and 16·1 per cent. in those with the apex beat at the normal level.

It is among the younger patients with raised apex that the greater frequency of a faint tricuspid murmur occurs. For if the cases who are over eighteen alone be compared, it is found that out of the 57 cases with an upward displacement of the apex there was a tricuspid murmur (well marked or faint) in 28·1 per cent. and among those with a normal apex beat in 28 per cent. of the cases.

Therefore, it is clear that there can be no causal relationship between the agencies producing the upward displacement of the apex and those causing the appearance of a systolic tricuspid murmur.

5. FAILURE OF LEFT VENTRICLE NOT ASSOCIATED WITH A RAISED APEX

The evidence all points to the fact that failure of the left ventricle does not play any part in causing the rise of the apex beat, for it is on the whole less frequent in these cases than in those with a normal apex beat.

As evidence on this point it is not possible to take outward displacement of the apex as a sign of failure of the left ventricle because the rotation of the heart which carries the apex up into the fourth interspace must at the same time swing it outwards external to the nipple line.

Evidence can only be sought therefore from noting the frequency of a mitral systolic murmur in the two groups of cases.

The figures are as follows :—

Among the 178 cases dealt with, a systolic mitral murmur was found to be present 38 times—i.e. in 21 per cent.; but of these it was only a faint murmur in 12 instances, thus there was a well-marked murmur in only 26, or 14·6 per cent. of the total number of cases.

Among the 85 cases with a raised apex beat, a mitral systolic murmur was present in 16 instances, or 18·8 per cent., and was well marked in only 12·9 per cent.; whereas among the 93 with a normal apex a murmur was present in 22, or 23·6 per cent., and well marked in 15, or 16 per cent.

The detailed figures are as follows :—

	With raised apex		With normal apex	
	No.	Mitral murmur	No.	Mitral murmur
Age 18 or under . .	28	2 = 7%	43	9 = 20·9%
Age 19 or over . .	57	14 = 24·5%	50	13 = 26%
	85	16 = 18·8%	93	22 = 23·6%
With well-marked murmur	—	11 = 12·9%	—	15 = 16%

It can therefore be said that failure of the left ventricle is not only *not* a factor in the causation of an upward displacement of the apex in the cases we are dealing with, but that its presence seems to render the occurrence of upward displacement less liable to occur.

CONCLUSIONS AS TO THE CAUSE OF UPWARD DISPLACEMENT OF THE APEX

Having now summarised the facts which the study of the 178 cases of anæmia has elicited, we are in a position to discuss the cause of the upward displacement of the apex.

Firstly, it is rather more frequent in older than in younger adolescents. Now, as has already been pointed out, in the younger patients, the dilatation of the right ventricle is more apt to be limited to the thin anterior part of the muscular wall near the pulmonary valves; whereas in older adolescents (owing presumably to the increasing strength of the fibrous tissues of the heart), there is more tendency of the ventricle wall to give way evenly.

1. Therefore, it can be asserted that the *general* yielding of the wall of the ventricle does appear to play some part in the causation of the rise of the apex beat, whereas the yielding of the so-called conus arteriosus, apart from general dilatation of the ventricle, does not.

2. Where the general dilatation of the ventricle is so marked that the right border of the heart is carried beyond its normal position, the apex beat is less likely to be found raised above its normal level than is the case where the right border of the heart is normal.

3. Upward displacement of the apex is less likely to occur where the left ventricle is becoming dilated than it is in those cases where the left side of the heart shows no signs of dilation.

Therefore, the conditions which tend to favour a rise of the apex beat appear to be a general dilatation of the anterior wall of the right ventricle combined with a normal right border of the heart and a left ventricle, which does not show any signs of enlargement.

A consideration of the physical conditions which these three factors bring about suggests that the main factor in the causation of the rise of the apex beat is such an extension of the right ventricle towards the left as to carry the anterior margin of the interventricular septum to the left of its normal situation.

We may therefore say that the apex is rotated up in those cases where there is a tendency to dilatation of the main cavity of the right ventricle without any yielding of its right border, and without any dilatation of the left ventricle. This statement implies that the main factor in the causation of the rotation is an increase in the size of the right ventricle towards the left, for our observations seem to show that there is no increase in the other directions. Its right boundary is normal, its posterior boundary—the interventricular septum—is normal, for the left ventricle is not enlarged. Its lower boundary is normal, limited by the diaphragm. Its upper border can be neglected in this connection, because the study of our cases shows clearly that upward dilatation occurs without any upward displacement of the apex. As regards forward increase the presence of the anterior chest wall seems to prevent the simple bulging forward of the heart, which would otherwise occur.

There only remains, therefore, dilatation of the right ventricle to the left.

It is, I think, demonstrable that a dilatation of the right ventricle in this direction may well cause a tilting upwards of the apex. To allow of increase in this direction it would be necessary for the left ventricle to be pushed outwards and backwards.

Now the most fixed point of the heart is its attachment to the diaphragm (where the inferior vena cava passes through that structure), and, as has been already pointed out, in these cases of dilatation of the right ventricle, there is no evidence of any dilatation of the right auricle, and therefore this fixation point is not interfered with, as it would be, were the tissues of the auricular wall stretched by dilatation of that chamber.

The attachment of the heart by its vessels to the structures which form the roots of the lungs constitutes a second fixation point, so far as rapidly acting and not too powerful distorting forces are concerned, and it is this fixation point with which we have now to deal.

The fact that the left ventricle is fixed by the connection between the aorta and pulmonary artery at the base of the heart and is free to move only at the apex, prevents it from making room for the right ventricle by simply moving away to the left.

The left ventricle will therefore act towards the right as if it were a rigid board swinging on a hinge placed at the base of the heart. If more room is needed to the right of this board the tip of the board will have to travel to the left, outwards, in the arc of a circle whose centre is the hinge.

This then, is the mechanism whereby the apex of the heart is carried upwards. If the left ventricle has to move away to the left to make room for an increase in the volume of the right, any movement to the left must be of the nature of a rotation, for the apex cannot increase its distance from the fixed point at the base.

The interventricular septum acts as the radius of a circle, and, on swinging to the left to allow of more room for the right ventricle, must carry the apex of the heart upwards in its attempt to carry it outwards.

This appears to be the true explanation as read from the clinical facts. It is not, perhaps, what might have at first sight have been expected to take place. The natural thing would have been for the ventricle to increase to the right of the sternum, as it does ordinarily when there is considerable dilatation of this chamber. Or else it would be expected simply to bulge forwards against the anterior chest wall and find room for itself by simply displacing the left ventricle a little backwards. This, too, does not appear to be the case, as shown by the clinical facts.

To summarise: The apex of the heart may be displaced upwards in cases of anæmic dilatation of the organ by means of a rise in the average level of the diaphragm upon which it rests, or by means of a simple downward extension of the right ventricle which raises the apex by pressure against the diaphragm.

Or, thirdly, the apex may be raised in consequence of the left ventricle being swung outwards and upwards (around its basal fixation point as centre) in order to allow of an increase in the right ventricle *towards the left.*

Another cause of upward displacement of the apex appears to be the simple shrinkage in the size of the heart, which is described at p. 50 as accompanying a diminution of the total volume of the blood in active circulation. Such an elevation of the apex beat is shown in figs. 42, &c.

The following case shows the rapidity with which a rise in the position of the heart may come on in an adolescent when the pulmonary circulation is interfered with by pneumonic consolidation.

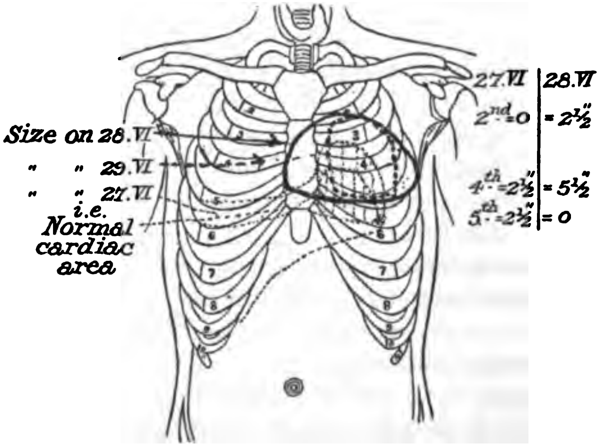


Fig. 129.

H. H., aged seventeen. Acute pneumonia. Rapid dilatation of heart with upward displacement.

H. H., aged seventeen. Taken ill, June 25, at 9 A.M., with abdominal pain and vomiting. On admission to hospital next day, he had severe pain in the right iliac region, the muscles on the right side of the abdomen were rigid, and he was supposed to have appendicitis. The pain was, as the result proved, really due to a right basal pleurisy which gave no signs at first. The lungs which were clear forty-eight hours after the onset of his illness, rapidly became consolidated, and by the evening of the second day the right lower lobe was affected, and on the third day the left apex, the left lower lobe, and half of the right lower lobe were consolidated. On this day (June 28), the cardiac dullness, which on the previous day was normal, was as follows : upper border at the second rib, left border at the nipple line in the second interspace, and in the fourth interspace it was at the mid-axillary line, 5½ inches from the sternum. The lower border of the heart was at the fifth rib. The right border of the heart was at the right edge of the sternum. Two days later, when the dilatation of the heart was less extreme, it assumed the state which is typical of the condition we are describing. Its upper border was at the second rib, and its lower border at the fifth. Its right border was 1 inch to the left of the sternum, and its left border was nearly an inch external to the left nipple line. At this time the whole of the left lung was consolidated and the lower half of the right lower lobe also (*vide* fig. 129).

But most cases of cardiac displacement are due to changes in the surrounding viscera.

II. Extrinsic Causes of Displacement

The heart may be pushed or pulled out of place by alterations in the volume of the lungs, or of the abdominal organs, or it may be displaced by the growth of tumours in the thorax or abdomen.

The displacement may take place in any of the six directions in which a body is free to move in this space of three dimensions which is the characteristic of our universe. I shall base my classification of cardiac displacements upon the direction of the displacement, and in the following order : (1) forwards ; (2) backwards ; (3) downwards ; (4) upwards ; (5) to the left ; (6) to the right.

1. Displacement Forwards. — The normal position of the heart is such that no considerable amount of forward displacement is possible, although aneurysm or tumour in the posterior mediastinum would cause rather more of the heart than normal to come into contact with the anterior chest wall.

2. Displacement Backwards.—This is of course frequent in emphysema, where it occurs in combination with displacement downwards, as a result of the over-distension of the lungs, and especially of the thin margins which overlie the heart. A certain amount of displacement backwards also frequently

results from compensatory enlargement of the right lung which accompanies disease confined to the left one.

Also, as was pointed out when reference was made to the smallness of the area of the cardiac dullness in cases of myocardial weakness in distensible hearts, a certain amount of the apparent smallness of the heart when the patient lies back may be due to the underfilled heart not being large enough to come into as close contact with the anterior chest wall as a full heart would.

It is possible also that there may be some backward displacement as well, but it is a point of no importance.

Cases showing this diminution of the cardiac dullness are given at pp. 118, 183, 196, &c.

3. Downward Displacement of the Heart Owing to Abnormal Lowness of the Diaphragm

Downward displacement of the diaphragm will result whenever the volume of the thoracic contents is considerably increased.

This is most commonly observable when there is a considerable accumulation of liquid—whether of serum or of pus—in either pleural cavity. The marked downward displacement of the liver in such cases is satisfactory evidence of this alteration in the level of the diaphragm. Another common cause of over-filling of the thorax is where the volume of the lungs is permanently increased as a result of their over-distension by frequent coughing in the condition known as emphysema. Then, as is well known, not only is the horizontal measurement of the chest increased giving it the classical 'barrel shape,' but its floor is also lowered, and the resonance of the lungs is found to extend to a lower level than normal. The lowness of the diaphragm is not only shown by percussion of the upper border of the liver, but also by the increased accessibility of the heart to palpation in the epigastrium.

Another common cause of downward displacement of the heart, due to the lowering of the diaphragm (upon which it rests), is not, I believe, so generally recognised as those already mentioned, and it must therefore be dealt with a little more fully.

In this case, the overfilling of the thorax is due to excess of the fluids normally present in it.

We have spoken of downward displacement from the abnormal presence of watery fluid in the pleura and from abnormal distension of the lungs by air, and we now have to speak of downward displacement of the diaphragm and heart by the persistent presence of an abnormal amount of blood in the thorax.

In such cases as severe mitral stenosis, where the heart muscle is strong, we know that there is chronic engorgement of the lungs with blood and also chronic over-distension of the great veins.

If a series of such cases be carefully examined it will be found that downward displacement of the diaphragm and heart occurs too frequently to be a mere coincidence.

In valvular disease with—what appears to be—a considerable dilatation of the heart towards the epigastrium and considerable downward enlargement of the liver, it is not infrequently possible to demonstrate that the epigastric pulsation must be in part due simply to downward displacement of the heart, and that part of what appears to be enlargement of the liver must also be simply downward displacement due to the lowness of the diaphragm.

The theoretical considerations in connection with the elevation of the diaphragm which occurs in cases where the amount of blood in the thorax is less than normal have already been dealt with in Essay II, p. 163.

The proposition here made is the simple converse of that; and the careful percussion of the liver and the auscultation and percussion of the heart in a series of cases where there is well-marked venous plethora (the result of chronic valvular disease with imperfect compensation) will clearly demonstrate the accuracy of what is here stated—namely, that chronic venous engorgement of the lungs and the thoracic veins does lead to a lowering of the diaphragm, and consequently to a downward displacement of the heart which rests upon it. For careful percussion and auscultation will show that the upper level of the liver dullness is often lower than normal in these cases, and that the upper border of the heart—as judged both by percussion and by the point of maximal intensity of the pulmonary second sound—is also lower than normal.

A slight case of this nature is given at p. 218, where the venous plethora was presumably due to overstrain of the heart apart from valvular disease.

The following is a well-marked case of this nature where the lowness of the whole heart was also confirmed by a radiograph.

Low Diaphragm due to Chronic Over-distension of the Lungs and Thoracic Veins with Blood

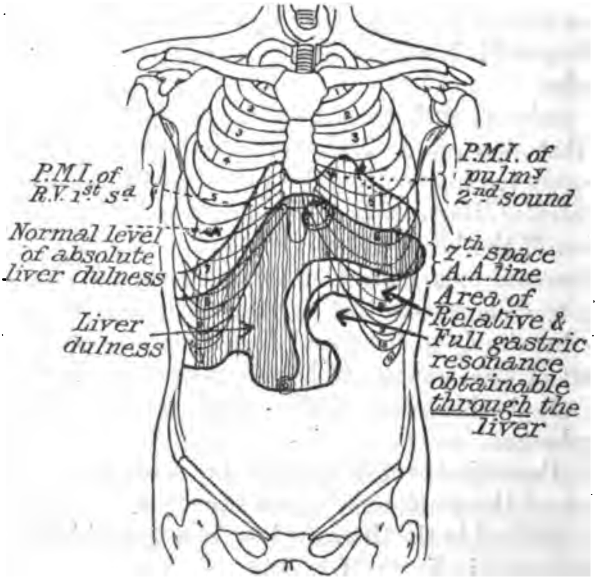
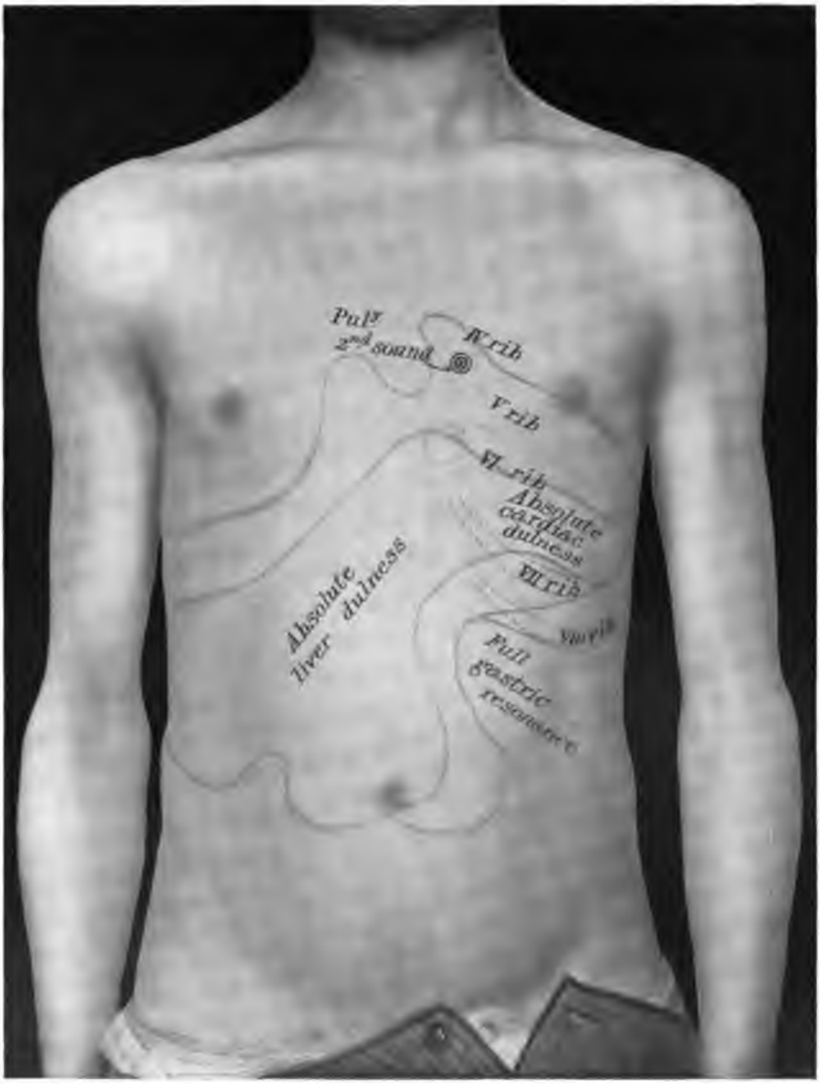

P.M.I. of
R.V. 1st sd
Normal level of absolute liver dulness
Liver dulness
P.M.I. of pulmy 2nd sound
7th space A.A. line
Area of Relative & Full gastric resonance obtainable through the liver

FIG. 130.

Arthur H. P., aged thirty. Mitral stenosis. Downward displacement of the heart by long continued chronic venous congestion.

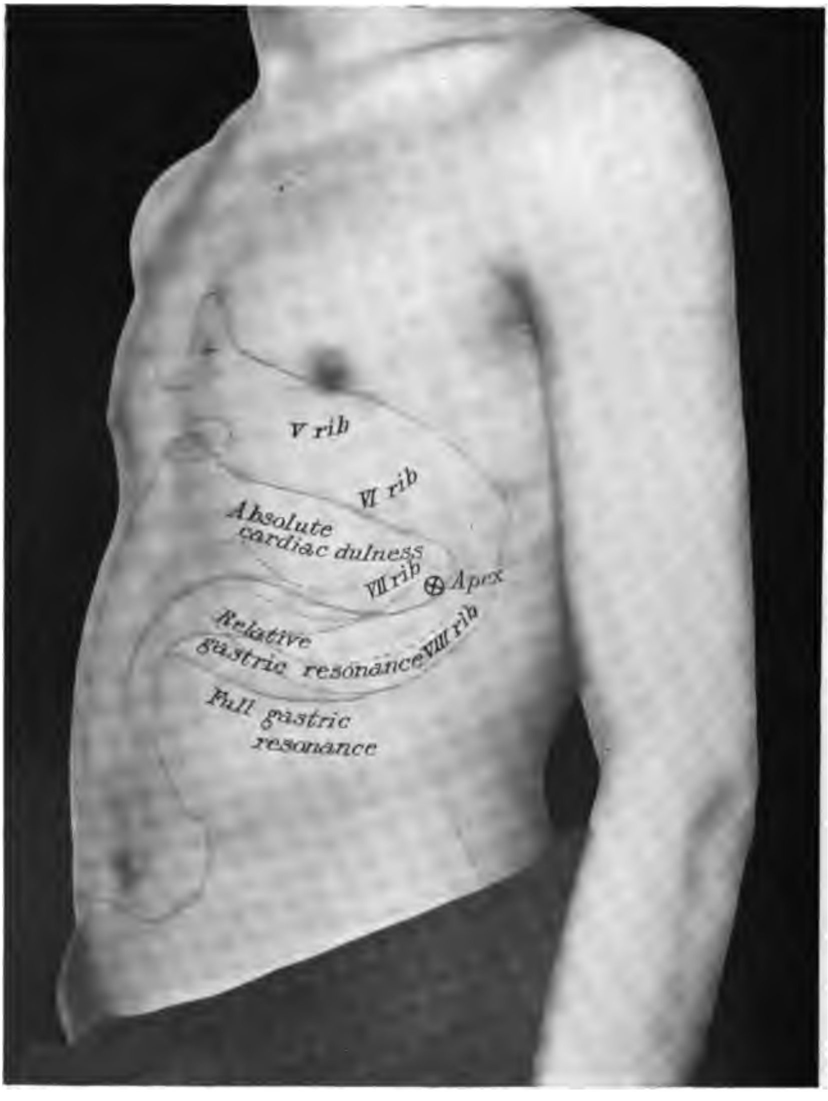
It is a most striking case, as will be seen by studying fig. 130 together with the actual photographs of the patient's chest. Previous to taking the photographs, the outline of the cardiac area and the liver dullness were marked on the chest in ink, and also the areas over which the pulmonary second sound and the cardiac murmur were audible, as well as the area over which the right ventricular impulse was best felt (*vide* Plates

PLATE IV



DOWNWARD DISPLACEMENT OF THE DIAPHRAGM AND HEART OWING TO CHRONIC OVER-DISTENSION OF THE THORACIC VISCERA WITH BLOOD.

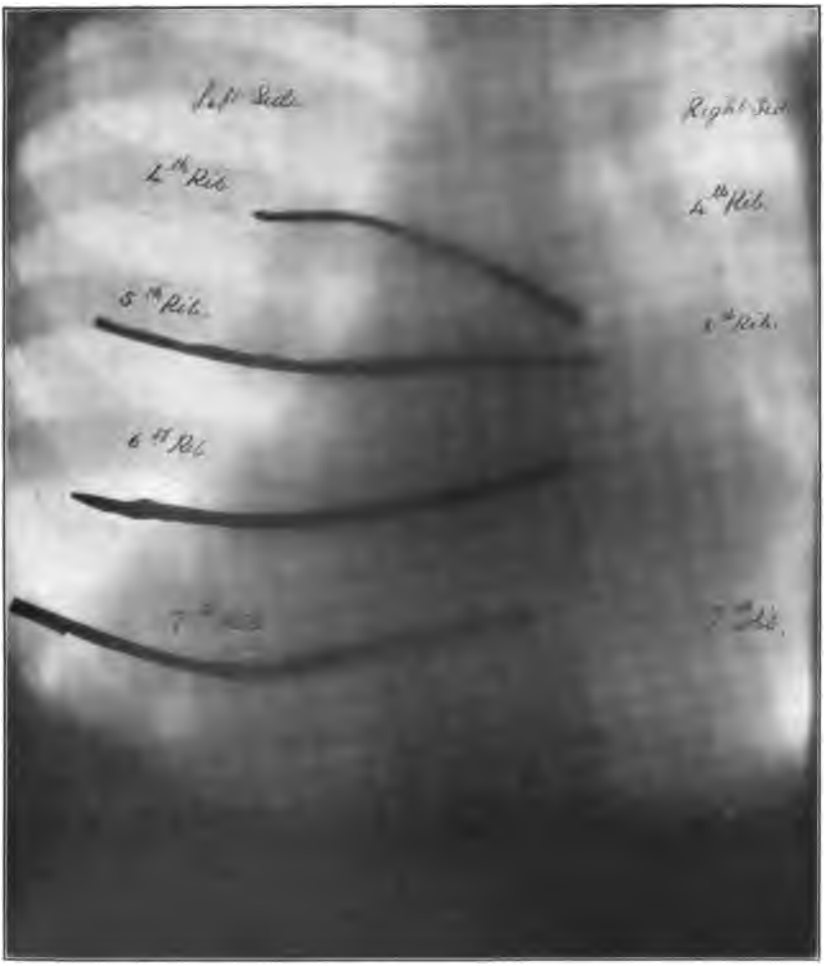
Arthur H. P., mitral stenosis. Photograph of the chest, showing the outline of the viscera as determined by percussion when recumbent. Note the close correspondence between the actual lines as drawn on the chest and the diagram shown in fig. 130. Note the lowness of the cardiac area, also the way in which the gastric resonance is obtainable through the liver owing to the increased conductivity to percussion vibrations which results from its over-distension with blood.



DOWNWARD DISPLACEMENT OF THE DIAPHRAGM AND HEART OWING TO CHRONIC OVER-DISTENSION OF THE THORACIC VISCERA WITH BLOOD.

Arthur H. P., mitral stenosis. Lateral view of the chest showing the outline of the viscera as determined by percussion, when recumbent. Note the downward and outward displacement of the heart.

PLATE VI



DOWNWARD DISPLACEMENT OF THE DIAPHRAGM AND HEART, THE RESULT OF CHRONIC OVER-DISTENSION OF THE THORACIC VISCERA WITH BLOOD.

Arthur H. P., mitral stenosis. Radiograph taken with the patient lying on his face. The position of the fourth, fifth, sixth, and seventh ribs anteriorly was marked by strips of lead.

Note that the main bulk of the heart is below the fifth rib, thus confirming the clinical observation that the pulmonary valves were below the fourth rib. Note the shadow of the dilated right auricle lying between the fifth and seventh ribs (instead of between the third and fifth), also the absence of shadow in the seventh right interspace showing the presence of lung at that level and the lowness of the diaphragm.

The outer ends of the lead strips marking the sixth and seventh ribs have slipped out of place and lie at too low a level.

IV and V). The displacement is also shown by the radiograph reproduced in Plate VI.

The patient was a man aged thirty, an engraver by trade, who had had 'rheumatic fever' three times before he was seven years old, and not at all since, although he had often suffered from rheumatic pains.

As a lad, he had no dyspnœa, and played football and hockey. His present cardiac incapacity began some two and a half years ago, with sudden very sharp pain in the left præcordium. He got better after some months, and was able once again to play cricket; but then a fall which dislocated his shoulder made him worse again, and he has been breathless ever since. For some months now, fresh symptoms have come on instead of the breathlessness. He is now prevented from exerting himself much by a sense of swelling in the epigastrium accompanied by palpitation, which forces him to rest until it passes off. Also his legs and arms and even his body go cold on exertion—even in warm weather. After he has walked a quarter of a mile or so, he often has to stop because of this coldness, and after resting five minutes or so his feet and legs go warm again. He has no leg pains on exertion (*vide* p. 469).

The examination of the heart showed he was suffering from mitral stenosis with evident signs of faulty compensation.

The cardiac area was, however, much lower than normal, and the lower margin of the lungs was also very greatly below the usual level—as reference to the illustrations will show.

The cardiac pulsation and dullness extended from the fourth rib down to the eighth rib, and the amount of cardiac dilatation was also shown by the fact that the cardiac area stretched from 1½ inches to the right of the sternum in the fourth interspace to the left anterior axillary line in the seventh.

At first sight it would be thought that the presence of the apex beat in the seventh interspace in the anterior axillary line would mean that the heart was enormously dilated. On noting the base of the heart, however, it was found that the cardiac dullness did not extend above the fourth rib, and that the point of maximal loudness of the pulmonary second sound was in the fourth left interspace instead of the second. This fact pointed towards downward displacement of the heart owing to descent of the diaphragm. On examining the liver dullness to find if there were any other evidence of a low diaphragm, strong confirmatory evidence was forthcoming, for the edge of the right lung came down to within one inch of the costal arch and crossed the eighth rib in the nipple line instead of the sixth. The relative dullness of the liver was at the seventh rib in the right nipple line instead of the fifth. Posteriorly, the same phenomenon was observable, for the lungs came down to below the twelfth rib on both sides.

There was nothing in the patient's history to explain the abnormal size of the lungs, for he could not take violent exercise and had never suffered from bronchial trouble. Once, four years ago, he had a cough brought on by exertion, but it subsided in a few days. Therefore, there was no reason to suspect the presence of emphysema ; and the probability is that the low diaphragm is a result of the chronic engorgement of the lungs and not a mere coincidence.

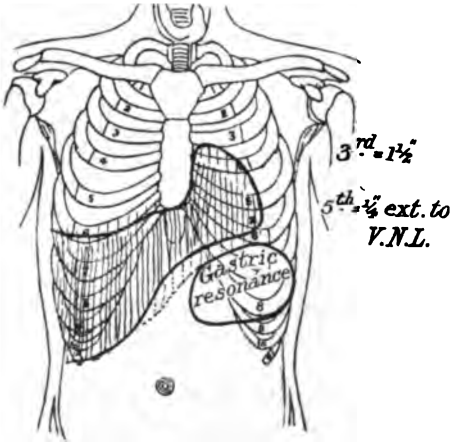


FIG. 131.

George G., December 1904. Mitral stenosis. Showing the size and position of the heart during twelve years 1892–1904, while compensation remained good and there was no evidence of congestion of the lungs.

Two figures are given of another case showing how much the level of the heart dropped in the course of seven years—namely, figs. 131 and 132.

The case was one of pure mitral stenosis with such an extreme degree of narrowing that even when compensation was at its best and the patient able to get about fairly well there was a very loud pre-systolic murmur and thrill as well as a very loud mid-diastolic mitral murmur. For twelve years, from 1892 till 1904, while the patient was comparatively well and able to work, the apex remained in the fifth interspace in about the nipple line. As a rule, during this period, there was no evidence of chronic venous congestion of the lungs.

Two drawings are given : one showing the size and position of the heart in December 1904 to have been normal except that the apex was just external to the nipple line. The second one shows the condition of the heart seven years later—namely, November 1911, when it was displaced downwards.

There are distinct evidences of displacement downwards, although the drop of the apex beat from just external to the nipple line in the fifth space, to the sixth interspace one inch external to the nipple line, might well be accounted for by simple dilatation and hypertrophy. The falling of the point of maximal loudness of the aortic and pulmonary second sounds, however, from the second to the fourth interspaces, is very suggestive of displacement, as is also the descent of the upper edge of the liver dullness from the sixth rib to the seventh. For some years prior to 1911, compensation had been failing.

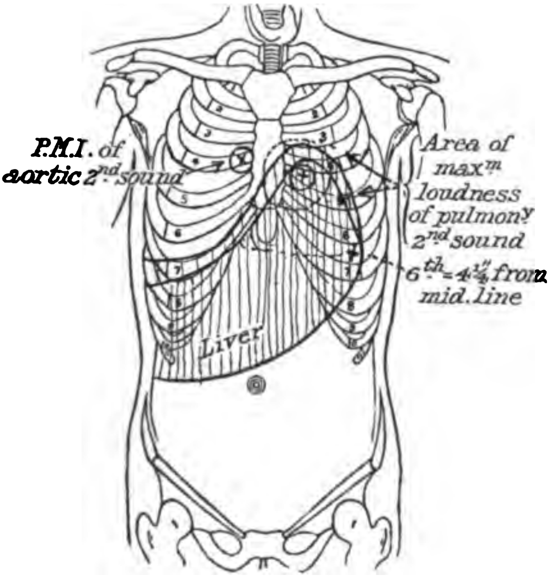


Fig. 132.

George G. Mitral stenosis, November 1911. Showing downward displacement of the heart and enlargement of the liver due to some years of chronic venous congestion from failing compensation.

Displacement of the heart downwards will also result from the growth of tumours in the thorax if of sufficient size—as in the case of endothelioma of the pleura or mediastinal lymphosarcoma.

4. Displacement of the Heart Upwards.—As the heart lies upon the central tendom of the diaphragm, anything which causes the ascent of that structure will raise the heart to a higher level in the thorax than normal. Anything increasing the intra-abdominal pressure sufficiently to raise the diaphragm will therefore cause upward displacement of the heart. This occurs in the case of ascites, or the abdominal distension of peritonitis, enteric fever, &c. More important from the clinical point of view is the upward displacement of the heart which is brought about by flatulent distension of the stomach, because this is very often seen in heart failure, as a consequence of faulty circulation, and the additional cardiac embarrassment caused thereby may, when life is trembling in the balance, turn the scale against recovery.

But the diaphragm does not only rise as a result of pressure from below, it may also undoubtedly be *drawn* upwards by a diminution in the volume of the intrathoracic contents. As to the nature of this diminution, I am not at present prepared to dogmatise.[1] It may sometimes be due mainly to a diminution in the size of the lungs owing to changes following upon alterations in the respiratory functions, as in the cases of chlorosis ; but it is in my opinion certainly due, sometimes, to a diminution of the total volume of the blood which the heart is able to put in circulation. For instance, in a case of sudden cardiac failure—such as from an influenzal affection of the heart—the diaphragm can be observed, in the course of a day or so, to rise to an inch or more above its normal position, carrying the lower border of the heart up to above the level of the fifth rib in, and internal to, the nipple line on the left side.

The following is an instance of such a case :—

A young man, aged about twenty-eight, in good health, was suddenly seized with severe abdominal pain of a colicky nature, accompanied by syncopal attacks, in the first of which his pulse rate fell to below 40 per minute. The heart remained very feeble, and some ten days or so after the onset of his illness, the cardiac dullness was greatly diminished in area, and its lower level did not reach below the fifth rib. Although there was not the least sign of any gastric distension, full gastric resonance was obtainable up to the fifth rib in and internal to the left nipple line, and in the middle line under the sternum full gastric resonance was obtainable up to the level of the fifth rib, and full pulmonary resonance above it. To the right the liver was one interspace higher than normal. As the heart got stronger, the liver and the stomach returned to their usual level, showing that the diaphragm, and with it the heart, had also returned to its normal position (see fig. 41, p. 183).

---

[1] The greater part of this essay was written in 1901. See *Birmingham Medical Review*, October 1901. Compare also Essay II.

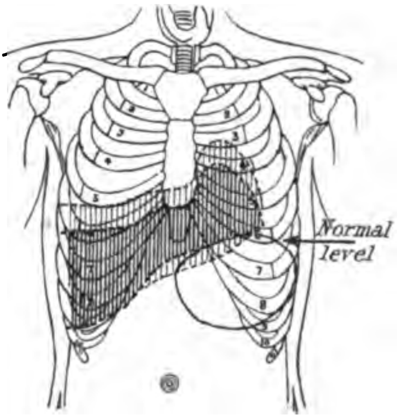


FIG. 133.

NORMAL PERCUSSION OF HEART, LIVER, AND STOMACH.

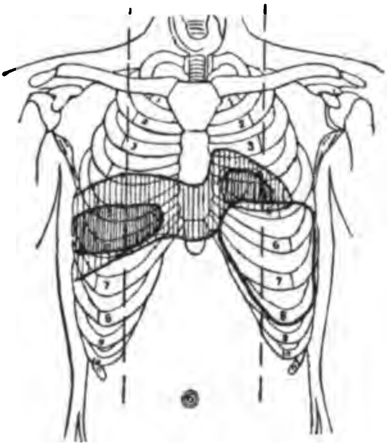


FIG. 134.

SHOWING DISPLACEMENT UPWARDS OF HEART, LIVER, AND STOMACH.

In both figures the close shading represents absolute dullness and the light shading relative dullness. The area of gastric resonance is outlined and left unshaded.

Another condition in which this upward displacement of the heart (due to the ascent of the diaphragm) is observable, is where it may reasonably be supposed that there is a diminution in the total volume of the blood in circulation. Thus I have seen it more than once in enteric fever accompanied by a hollowing of the abdomen and a shrinkage of the limbs and face—evidently pointing to a diminution of the circulating fluids of the body, from failure of absorption of nourishment.

A very marked case, of which I now give some particulars, occurred in a young woman under treatment for gastric ulcer with a suspicion of impending perforation. The diminution in the supply of nourishment, necessitated by rectal feeding, brought about the following condition (*vide* fig. 134).

A. V., aged twenty-one, 'was fed mainly by the bowel until three or four days ago. The gastric resonance to-day reaches up to the upper border of the fifth rib in and internal to the left nipple line, and there is no dullness at all in the fifth interspace. The lower border of the liver in the middle line does not reach as low as the tip of the xiphoid cartilage, and in the right nipple line the relative dullness of the liver commences at the fourth rib, and the absolute dullness at the fifth rib, while the lower edge of the liver does not seem to reach lower than the sixth interspace as there is full intestinal resonance below this level. In the mid-axillary line intestinal resonance reaches as high as the eighth interspace, and the lower edge of the lung only comes down to the lower border of the sixth rib.' The condition is shown in fig. 134, and a normal chest is shown in fig. 133 for comparison with it.

UPWARD DISPLACEMENT OF THE HEART BY HIGH DIAPHRAGM

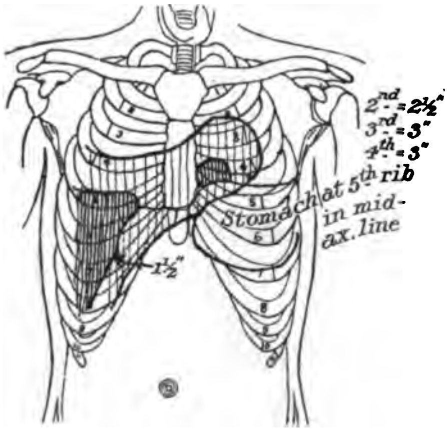


FIG. 135.

Nurse T., aged thirty-one, October 30, 1913. Upward displacement of the heart by high diaphragm due to malnutrition accompanying mucous colitis. No murmurs.

Fig. 135 also shows a well-marked case of this nature occurring in a lady aged thirty-one, who, because of pain, had greatly limited the amount of food she took, but had at the same time continued to live an active life. The chief symptom besides her pain was a feeling of undue weariness. Considering the condition of her circulation such a symptom could easily be understood (see also Essay II).

It is presumably possible for the apex to be raised by other extrinsic agencies, such as by traction by pleural adhesions when the pericardium is adherent; but such are not often met with and are of little clinical importance.

Lateral Displacement of the Heart

Before taking up the question of lateral displacement of the heart, it will be well to say a few words as to the mobility of the heart. Careful clinical observation seems to show clearly that it is not correct to regard the heart as being 'fixed' in its position by the fibrous tissue which surrounds the aorta and the roots of the lungs, nor yet by the attachment of the inferior cava to the diaphragm.

These points of 'fixation' must only be regarded as relative and not absolute, for, even in adult life (when the fibrous tissues may be regarded as having attained to a full degree of firmness) the heart and the tissues of the mediastinum can shift from their position in the middle line of the body and travel either to the left or to the right, if one or other of the lungs collapses, and the other becomes compensatorily enlarged, and the apex of a heart of normal size may be found as far from its normal situation as the left posterior axillary line or the right anterior axillary line.

In adolescence, owing to the softness and immaturity of the fibrous tissues, these changes in the position of the heart may take place with almost unbelievable rapidity, as in the following case where (owing to the sudden collapse of an inflamed left lung, as a result of vaccine treatment) the heart in the course of less than ten days shifted over some two inches to the left of its former position, so that its right border was almost in the left nipple line and its apex in the axilla. The greater part of this movement, moreover, took place in the course of only three or four days.

The following are the details of the case.

The patient, Lily B., eighteen years of age, was admitted to the General Hospital, under Mr. Haslam (to whom I am indebted for permission to refer to the case), in January 1908, suffering from a septic condition of the right kidney.

In spite of excision of the kidney, pyæmia developed. The temperature rose to 103·6° F., and an extensive amount of septic pneumonia developed—both lungs being affected. The left one was most involved, and there were many areas of dullness and tubular breathing scattered throughout the whole of the lungs with very numerous liquid râles. The lower part of the right lower lobe was similarly affected. It

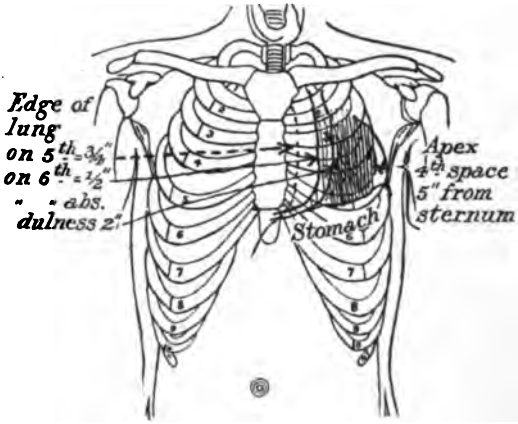


Fig. 136.

Lily B., aged eighteen. Rapid displacement of the heart to the left by collapse of the left lung on the arrest of pyæmic pneumonia. On March 5, edge of right lung was ¾ inch to left of the sternum. On March 6, edge of right lung was 2 inches to left of the sternum. Also on this date there was full lung resonance for 1½ inches to the left of the sternum, and gastric resonance in the fifth space to the anterior axillary line.

was certainly a case of septic broncho-pneumonia, and the prognosis was very grave indeed as pyæmic abscesses were—almost certainly—on the point of forming throughout the lung. At my suggestion, an exploratory puncture of the lung was made, and it was found that the infection was streptococcal in nature, and an autogenous vaccine was prepared. A first dose of 10 million was given on March 1, and a second on March 3, together with 100 million staphylococci obtained from the operation wound. A striking improvement followed the first injection of vaccine, and by the fifth of March there was a great change in the condition of the left lung. The upper and middle lobe on the right side, where there had been some patches of consolidation, were now quite clear, but there was still some consolidation with tubular breathing at the base of this lung. As regards the left, the whole of this lung was now uniformly consolidated, and there were numerous moist râles. But this increased consolidation was shown not to be due to fresh pneumonic infiltration by the fact that the heart was being drawn and pushed towards this side, for the right lung now crossed over and its edge could be detected ½ inch to the left of the sternum. This condensation and shrinkage of the left lung progressed so rapidly that next day the edge of the right lung was fully 2 inches to the left of the sternum, during inspiration, from the first rib down to the fifth, and for 1½ inches to the left of the sternum there was no cardiac dullness at all. Both lungs also were clearing very satisfactorily, and there were hardly any râles to be heard. Next day, March 7, the heart had moved still farther towards the left, and its right border was nearly 2 inches from the sternal edge, and edge of the right lung was nearly in the left nipple line—i.e. some 2¼ inches to the left of the sternum. The apex of the heart was nearly in the anterior axillary line—more than 2 inches to the left of the position it had occupied three or four days previously. It was now 4¾ inches from the sternum, or 5¼ from the middle line (see fig. 136).

The reason for this sudden movement of the heart was what we may call the collapse of the left lung due to the sudden cessation of the inflammatory process brought about by the vaccine treatment.

In the course of a few weeks the left lung re-expanded and the heart returned to its normal situation.

The case has been published by Dr. Wynn, in its bacteriological aspect, as he had charge of the vaccine treatment.

In this girl, therefore, the whole heart shifted over fully 2 inches to the left of its previous position in the course of a few days only, and with such a case as this before us, we cannot speak of the heart being ' fixed ' in its normal position unless we use the term as a relative one only.

We now have to deal with—

5. DISPLACEMENT OF THE HEART TO THE LEFT.—Displacement of the heart to one side or the other is of extremely common occurrence in diseases of the lungs and pleura, and the presence and amount of the displacement is often of the greatest clinical value from a diagnostic point of view.

I need not point to the well-known displacement of the heart which occurs in extreme pleuritic effusion or in empyema. Its significance has long been recognised and appreciated.

In these cases the heart is pushed out of place by the pressure of the accumulated fluid. But, as in the last case, the heart may not only be pushed out of place by excess of pressure on one side of the thorax, but it may also be drawn out of place by the traction of a lung which is shrinking and becoming fibrosed as a result of chronic inflammation, or from other cause, or by the traction exerted by the absorption of pleuritic fluid when the lung is too much damaged to expand again. It is possible to produce a far greater amount of cardiac displacement in this way by traction of one lung (combined with a compensatory enlargement of the other) than in the first-mentioned way, by means of pressure.

It may be of interest not only to give some well-marked cases, such as that figured on p. 167 and that which now follows, but also some illustrating the value to the clinician of this cardiac displacement.

It would be easy to give many cases where great displacement of the heart to the left resulted from the cicatricial contraction of a lung that has healed after some inflammation, but the following case only will be given :—

Miss W., aged about twenty-six, was brought to me by her doctor on account of cardiac dilatation, because she was rather breathless and the heart's apex was in the axilla. On examining the chest it was found that the heart's apex was external to the anterior axillary line, and the cardiac dullness and pulsation reached nearly to the anterior axillary line in the third interspace. But on percussing out the right border of the heart it was found to be nearly in the *left* nipple line, and that the right lung crossed to about 2 inches to the left of the sternum. On examining the chest it was evident that the left lower lobe was almost completely fibrosed, and the right lung crossed over the middle line to the extent of 1½ inches or more. There was no history of illness or of expectoration, and the destructive process—a tubercular one—had gone on to this extent without the knowledge of the patient or the doctor who had seen her occasionally.

Cases showing such an extreme degree of displacement are of interest clinically, because they show the completeness of nature's efforts to cure by the absorption and cicatricial contraction of the diseased lung tissue. They have not, however, the same clinical and diagnostic importance which the slighter cases have, where the recognition of a slight amount of cardiac displacement may throw important light upon the diagnosis and treatment of the case.

The following is such a case—

Charlotte D., aged forty-four, admitted to the General Hospital with left pleural effusion. Her doctor had tapped her chest three weeks previously and drawn off clear serum, but it had apparently reaccumulated, and he therefore sent her to the hospital for further treatment. The left base was dull with diminished breath sounds, &c., as far up as the angle of the scapula, and exploratory puncture showed the presence of serum in the left pleura. The question now arose as to the need for again tapping the pleura, the breathing being considerably embarrassed, and the amount of dullness suggesting a not inconsiderable pleuritic effusion. On percussing the heart, however, it was found that the right border of the cardiac dullness did not reach the left edge of the sternum, because the resonance of the right lung crossed over to the left of the middle line as far as midway between the sternal edge and the left nipple line. The left border of the heart was also displaced one inch or so to the left of its normal position. From this slight displacement of the heart *towards* the pleural effusion it was evident that absorption was going on as fast as possible, and that there was already what might almost be called a negative pressure in the left pleural cavity. The reason for the non-absorption of the fluid was the inability of the lung to expand, and further tapping could do no good. The case was treated by respiratory exercises with a view to aiding the expansion of the lung with a satisfactory result.

Another case illustrating the value of a careful mapping out of the right border of the heart is that shown in fig. 137.

It is the case of a man, aged fifty-eight, who fractured a rib on the left side, some three weeks prior to his admission to my wards for pleuritic effusion on the injured side. On admission, there was dullness and absent breath sounds up to the third rib on the left side. The cardiac dullness reached to the edge of the sternum, showing little or no cardiac displacement. This was not to be wondered at in a man of fifty-eight, whose fibrous tissues must naturally be fairly rigid. Next day, 60 ounces of blood-stained serum were removed from the left pleural cavity, and afterwards it was found that the left lung remained rather dull and that the right lung crossed over to the left of the sternum for 1½ inches in the second left space, and ¾ inch in the third space, showing some displacement of the heart towards the affected lung. On examining the lower border of the right lung it was found to be displaced downwards (as shown in fig. 128), as was also the liver.

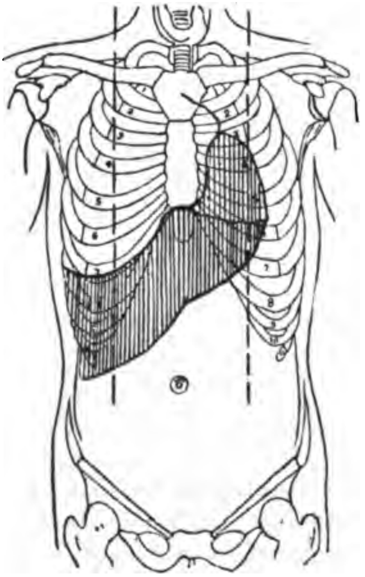


FIG. 137.
DISPLACEMENT OF HEART TO THE LEFT AND THE LIVER DOWNWARDS.

Now this patient gave a history of having had no previous illness; but, taking into consideration his age and the probable rigidity of his tissues, it was not credible that a pleurisy with effusion, lasting at most two or three weeks, could have caused the displacement of his heart and liver already described. Consequently, in spite of his statements to the contrary, it was certain that the unexpanded condition of the left lung dated back many months, if not years, and was not due to the traumatic pleurisy which followed the accident.

Careful questioning elicited the fact that he had been rather short of breath for a long time, and brought out a history pointing to a very long-standing pleurisy of the left side. Here the careful search for a slight degree of cardiac displacement led to the making of a correct diagnosis. The evidence given by the slight displacement of the heart would have been most important if an action for damages had been brought by the patient.

6. Displacement of the Heart to the Right.—In the cases of lateral displacement to which I have referred, the displacement of the heart has been to the left, but precisely similar help in diagnosis can be obtained where the disease has involved the right side, except for the fact that the heart does not seem quite so free to move towards the right side as towards the left, and cases of displacement to the right seem to be less frequent than those to the left (see also p. 381).

Displacement of the Heart to the Right by Gradual Collapse of the Right Lung

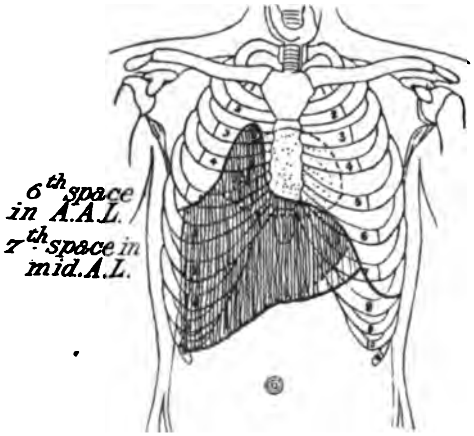


Fig. 138.

George B., aged thirty-five, October 20, 1904. Showing displacement due to fibrosis of the right lung.

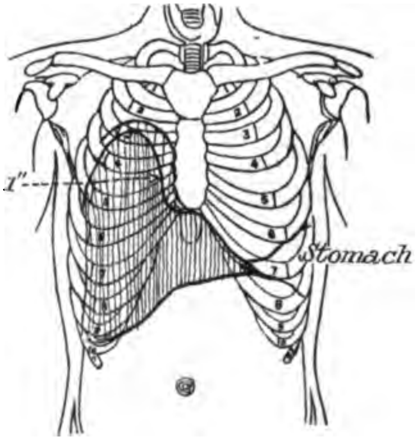


Fig. 139.

George B., March 30, 1906. Heart more displaced to the right. Edge of left lung 2 inches to right of the middle line.

The following is an extreme case of displacement of the heart to the right :—

G. B., a man about thirty-five, was seen by me first in October 1904. He was suffering from phthisis involving the right lung only. There was such marked retraction of the diseased lung with expansion of the healthy one that the cardiac dullness and pulsation was displaced to the right of the sternum, and the resonance of the left lung crossed the middle line, and during ordinary respiration was to be detected nearly 1 inch to the right of the sternum. The right border of the heart was close to the right nipple line in the fourth right interspace, as shown in fig. 138.

During the next two years extensive fibrosis took place, greatly reducing the size of the right lung (*vide* fig. 139).

By March 1907, the remains of the right lung occupied but a very small area in the upper and posterior part of the axilla and at the back.

In front, the full resonance of the right lung crossed well to the right of the sternum, and the edge of this lung reached to beyond the right nipple line in the first and second interspaces and was in the nipple line in the third interspace.

The cardiac dullness and pulsation began just to the left of the sternum in the fourth interspace and extended to nearly the mid-axillary line (*vide* fig. 140).

Posteriorly, the left lung crossed over the middle line to the extent of some 1½ or 2 inches.

This man died in March 1907 from amyloid disease of the kidneys, and the post-mortem examination confirmed the clinical observations as to the position of the heart and the extremely contracted state of the right lung. It was little more than the size of the hand and some 1½ or 2 inches thick, and consisted simply of fibrous tissue and dilated bronchial tubes or healed tubercular cavities.

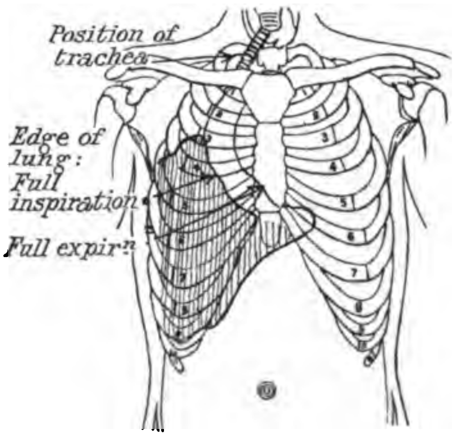


FIG. 140.

George B., March 1907. Heart in right axilla. Edge of left lung crosses over the right nipple line.

The heart lay on the right side, but most unfortunately I was not present at the post-mortem; and in spite of the unusual nature of the case no notes were recorded as to the relative positions occupied by the various parts of the heart.

In spite of this extreme displacement, the only murmur present was a moderately loud and localised systolic murmur audible about the right nipple line in the fourth and fifth interspaces.

Before leaving this subject, I will briefly refer to a case illustrating a somewhat less frequent cause of displacement.

The patient was an old man suffering from bronchitic symptoms. Examination of his chest showed the fact that his heart was much displaced to the right, and its right limit of pulsation was in the right nipple line. The action of the upper part of the right lung was very defective, and it was evident that there was a considerable amount of fibrosis of it. This shrinking of the right lung, coupled with the compensatory enlargement of the left lung, was the cause of the displacement of the heart. The fibrosis of the right lung was due to the infiltration of the root of the lung by a malignant tumour.

The man lived some months, and before his death the tumour had increased so much in size that its growth more than made up for the destruction of the lung, and the heart was pushed back into its normal position. This was verified at the post-mortem examination.

Altered Relationships of the Heart when Displaced

When the heart is pushed or drawn out of its normal position, there is often a certain amount of rotation also, and I propose in conclusion briefly to discuss this subject.

The fact that the heart rotates when moved in certain directions shows that certain of its attachments are more fixed than others, and it will help the consideration of the subject to summarise briefly its points of attachment and their relative fixity.

Generally speaking, the ventricles of the heart are most free to move, and the auricles the most fixed. But to understand the changes which take place, a more detailed view is necessary.

The right auricle is fixed to the diaphragm by the inferior cava.

The right auricle is fixed to the deep fascia of the neck, and the root of the lung by the superior cava, &c.

The left auricle is fixed to the root of the lungs by the pulmonary veins.

The upper anterior part of the right ventricle is fixed to the anterior part of the root of the left lung by the left branch of the pulmonary artery.

The upper anterior part of the right ventricle is fixed to the posterior part of the root of the right lung by the right branch of the pulmonary artery.

The upper central part of the base of the heart is fixed to the fascia of neck, &c., by the aorta.

Of these, the attachments of the aorta and superior vena cava are farthest from the heart and of less value than the others in preventing, or interfering with, cardiac displacement.

The attachments to the root of either lung are, on the other hand, short, and will greatly tend to hinder displacement of the organ, *provided* that the roots of the lungs are not themselves tending to move.

Now the attachment of the right auricle to the diaphragm by means of the inferior cava is the shortest of all the above-mentioned points of attachment, and the fibrous portion of the diaphragm to which it is attached is also, except for the normal range of upward and downward movement decidedly a fixed point. I believe this point, where the right auricle is practically fixed to the diaphragm, is to be regarded as the pivot around which the heart turns in the various displacements to which it is subject. In the lesser degrees of displacement, the fixation of the left auricle (which lies posteriorly) to the roots of the two lungs, is also a fixed point, so long as the roots of the lungs themselves remain fixed. In the extreme cases, however, where collapse of one or other lung is accompanied by great increase in size of the other, there is good reason to believe that the median position of the root of the lungs does not remain unchanged, but that the structures of the posterior mediastinum are moved over *en masse* by the shrinkage of the one lung and the increase in size of the other.

When this movement of the whole mediastinum takes place—as it seems to do very frequently in these cases—much rotation of the heart will not take place, and the only attachment likely to cause any alteration in the relative position of its chambers will be the fixation of the inferior cava to the diaphragm in cases where the heart is shifted to the right.

In any well-marked case it is easy to demonstrate the movement of the mediastinal tissues by noting the situation in which the lower end of the trachea enters the thorax. It is not uncommon to find it wholly to the right or left of the middle line, and in the case illustrated by fig. 140 the left side of the trachea was under the left border of the right sterno-mastoid.

A similar amount of displacement may also be seen on the left side.

This movement of the root of the lung is also suggested by the way in which the resonance of one lung will cross over the middle line posteriorly.

Over the lower part of the chest, where we often find the crossing over from one side to the other of dullness due to fluid, or of resonance due to compensatory enlargement, this crossing over has not the same significance, as it is simply due to the displacement of the membranous division between the two halves of the thorax when the pressure on one side exceeds that on the other.[1] It is, however, a phenomenon of very great diagnostic value.

With regard to the presence of rotation of the heart on itself when displaced upwards or downwards, there does not seem to be any reason to expect that such should occur, nor is there any clinical evidence of such rotation.

In displacement to the right, however, the case is different, and the changes the heart undergoes seem somewhat complicated. The following is a summary of a case illustrating this :—

T. F., aged forty-three, suffering from bronchitic symptoms. Physical examination of the chest showed that there was very considerable fibrosis of the right lower lobe, with some excavation. The upper part of the right lung and the whole of the left were healthy. The heart was drawn over to the right, and the cardiac dullness and pulsation were mainly to the right of the sternum. The cardiac and hepatic dullness were as shown in the diagram (fig. 141).

An auricle was pulsating in the right nipple line (over the area which is dotted in fig. 141), and this must have been the right auricle. There was ventricular pulsation above and to the left of this area, as far as the third interspace midway to the right nipple line. Over the upper part of this area the second sound was well heard, and its area of maximum loudness was in the third and fourth right interspaces, as shown in fig. 132. This must have been the right ventricle, and the area of maximal loudness of the second sound showed the situation of its conus arteriosus and the origin of the pulmonary artery to be some 2 to 3 inches to the right of the usual position. There was a little cardiac dullness to the left of the sternum in the fourth and fifth interspaces, with faint pulsation and almost inaudible heart sounds. This meant that the left ventricle was not as near the chest wall as normal.

---

[1] Where dullness due to pleuritic effusion crosses over, this area is sometimes called 'Grocco's triangle.'

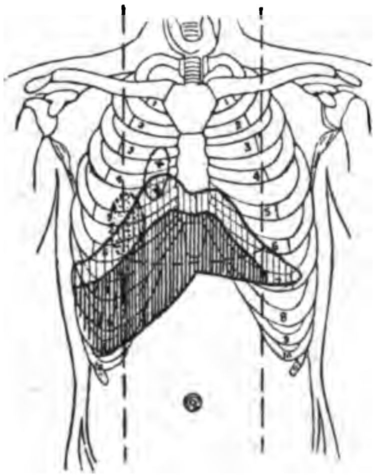


Fig. 141.

Displacement of Heart to the Right.

Dotted area = auricular pulsation.
× = area of maximum loudness of second sound.

The explanation of this condition is as follows, and it is a type of what may be expected in displacement of the heart to the right by shrinking of the lower part of the right lung :—

The traction of the contracted lung takes place through the branches of the pulmonary artery and vein which go to the affected lung.

Now the right branch of the pulmonary artery takes origin from the front of the heart, but passes behind the aorta to get to the right lung. Traction in this artery will therefore tend to rotate the heart about an axis running through the two relatively fixed points—namely, the aorta and the base of the right auricle where the inferior vena cava passes through the diaphragm. This rotation would bring the right border of the right auricle forwards into closer contact with the chest, and make the left ventricle and the apex of the heart recede from the chest wall just as was shown to be the case by the physical examination of the patient.

A similar effect will be produced by the traction on the pulmonary veins, running as they do from the diseased lung to the left auricle posteriorly.

As regards the displacement of the heart to the left by retraction of the left lung. In this case there is but little rotation, as the vessels pulled upon arise from the left side of the heart, and the fixed points are on the opposite side.

There is, however, sometimes a slight degree of rotation, and the left ventricle and auricle come to lie rather more anterior, and the right auricle more posterior, than would be the case were the heart simply pushed over as a whole.

As to the presence of murmurs. This is a somewhat complicated question, and one that is by no means settled at the present day. I am glad, however, to be able to make a clear statement on the subject—namely, that it is possible to have the most extreme degrees of displacement, either to the right or to the left, without any murmurs at all. This also holds true of the displacement which accompanies dilatation of the right ventricle, as described on p. 360, for in this case there was an entire absence of murmurs. If, however, these same displacements occur in anæmic individuals, there may be extremely loud murmurs, into which I cannot go in this paper. There is certainly no characteristic murmur in these cases, and I have often, I must confess, felt surprised at the absence of murmurs in cases of extreme cardiac displacement.

This absence of murmurs, even in extreme cases of displacement, shows that the attachments of the heart to the chest wall are nowhere rigid enough, as a rule, to produce kinking or compression of the large vessels, such as would be the case were, for instance, the roots of the lungs immovably fixed to the spine.

It also helps to explain the slightness of the cardiac symptoms which are noticeable in a simple case of cardiac displacement, even when extreme in degree—more especially when such displacement is towards the left. In fact, the heart seems capable of doing its work as easily when lying in the left axilla as when it is in its normal situation.

I cannot, therefore, say that there are any special symptoms characteristic of cardiac displacement, or that any special treatment is necessary.

PART III.—ESSAYS DEALING WITH VALVULAR DISEASE, ESPECIALLY IN RELATION TO VENTRICULAR EXPANSION.

ESSAY IX.—ON THE NATURE OF THE EXPANSION PHASE OF THE CARDIAC CYCLE AND THE PHENOMENA ASSOCIATED WITH IT[1]

[This essay is mainly a summary of what is brought forward in the three following essays with regard to the muscular expansion of the ventricles.]

THE object of this paper is to advocate the view that the ventricles of the heart are filled by their own muscular action and not by the auricular contraction, and to show how this theory suffices to explain the various phenomena observable in valvular disease of the heart.

It is more than twenty years since dissatisfaction was openly expressed with the old theory as to the forces which fill the ventricles, and the belief has since been gaining ground that the muscular fibres of the heart are able, not only to develop force by their longitudinal contraction, but also, by an act of longitudinal muscular expansion,[2] to enlarge the cavity of the heart so as to develop force in the reverse direction. In other words, the belief is surely gaining ground that the heart is a double-acting pump, filling its chambers by its own back stroke, and that the ventricles are not dependent upon the auricles, or other outside agency, so far as the reception of the main part of their charge is concerned.

It is, after all, only natural to believe that such a perfect pump as the heart is, should not be inferior, in its mechanism, to the pumps which the wit of man devises.

It was twenty-four years ago that this theory was first brought under my notice by Dr. Lockhart Gibson, then of Edinburgh and now of Brisbane, who pointed out the support which the experiments of the late Professor Rutherford gave to this view, by his demonstration that after receiving a minimal galvanic stimulus, a striped muscular fibre would, after contraction, *suddenly* return towards its former length, and that the force, developed by this sudden expansion, was approximately two-fifths of that developed during contraction.

---

[1] See *The Practitioner* for November 1907.

[2] Due probably to transverse contraction, according to Sir Lauder Brunton.

If these observations of Professor Rutherford had been generally accepted, the theory of the expansion of the heart muscle would have rested on a sure basis. There seems, however, to be some doubt as to the true interpretation of his experiments, and, therefore, in the absence of a physiological basis (apart from the fact that a transient negative phase is demonstrable in the intraventricular pressure curve), we have to depend upon clinical evidence. Clinical evidence is, however, not easy to obtain, owing to the nature of the problem.

Much can be done by the careful study of the heart's movements in health and disease by means of the cardiograph, but the time required to make careful observations, and then to measure and correlate the tracings, is very great, for very many tracings must be taken from all possible parts of the cardiac area, and from the large arteries and veins, before the interpretation of the cardiograms is possible with any degree of certainty.

As a result of very numerous observations during the last sixteen years, I am certain that the cardiograph shows conclusively that the phase of ventricular contraction *is* followed by an active expansion movement.

CARDIOGRAPHIC EVIDENCE

1. As is well known, the cardiac muscle does not at once relax after the closure of the semilunar valves, and, if the time of occurrence of the second sound is carefully recorded on the cardiogram by an electric signal, this can be clearly demonstrated in tracings, taken at the apex or over the centre of the heart. Thus the cardiograph shows that the heart muscle remains *hard*, and therefore *active*, for an appreciable time after the end of the systole, and relaxation does not immediately follow the end of the systole as marked, by the closure of the semilunar valves.[1]

---

[1] Essay X, p. 403.

2. Further, the cardiograph shows quite clearly that, during this post-systolic activity, the heart muscle does not remain *contracted*, for the size of the ventricle is rapidly increasing. This can be demonstrated, if a series of tracings is taken from one side of the central axis of an enlarged heart, where many different tracings are obtainable. Such tracings clearly show a fall, due to the diminution in the size of the ventricle as the blood is expelled from it; and following this fall is a sudden rise, pointing to the sudden return of the ventricle towards the size it was at the commencement of systole.

A record of the time of occurrence of the second sound, upon this type of cardiogram, shows that the maximal diastolic rise takes place immediately after the occurrence of the second sound of the heart.

I do not here refer to ordinary inverted tracings—because these, when they are not due to faulty manipulation, are not very trustworthy—but to the cardiograms showing a systolic rise, and then a fall followed by another rise (*vide* Plates VII and XII).

The fact that this diastolic increase in the size of the heart has power to lift the lever of the cardiograph against a pressure of some two or three ounces, or more, shows that it cannot be merely due to the elastic recoil of the *relaxed* muscle, neither can it be due, as has been suggested, to the inrush of the blood, which has accumulated in the auricle during the time of closure of the mitral valve. The mitral valve only remains closed for less than half of the cardiac cycle, the auricle cannot, therefore, be much more than half full when the mitral valve opens, and there can therefore be no inrush of blood under pressure from the auricle into the ventricle at this point of the cardiac cycle. Study of the cardiogram in dilatation of the ventricle hence appears to prove that true muscular expansion follows muscular contraction.

EXPANSION PHASE OF THE CARDIAC CYCLE

This period of expansion of the muscular fibres appears to be a definite and well-defined phase of the cardiac cycle. Both cardiographically and clinically, its duration is well defined, and careful measurements of the portion of the cardiogram representing this phase, as well as measurements of records of the cardiac sounds which mark its duration, show its length to be about one-tenth of a second. Moreover, its length does not seem to vary, as do the other phases of the cardiac cycle; but, whether the heart is beating slowly or rapidly, it still has a duration of one-tenth of a second.[1]

We must, therefore, alter our conception of the cardiac cycle, and insert in it a phase of expansion, thus :—

Ventricular contraction lasting $\frac{3}{10}$ of a second.
,, expansion ,, $\frac{1}{10}$,, ,,
,, relaxation ,, $\frac{4}{10}$,, ,,

The Function of the Auricles

This being so, certain other points arise in connection with the function of the auricles.

If the size of the mitral orifice is noted in relation to the cubic content of the left auricle, it must, I think, be acknowledged that, under powerful suction, the contents of this chamber would be immediately drawn into the ventricle, although the aspiration lasted only one-tenth of a second, and that therefore the auricle would be empty by the time relaxation of the ventricle set in. According to the old theory, the auricle also empties itself during its systole. If this were so, the auricle would be twice emptied in each cycle.

Now, during any particular cycle, as the flow of blood from the pulmonary arteries into the auricle is more or less regular, this double emptying of the auricle should result in its never having to hold the full charge of blood. In other words, if the heart throws 3 ounces of blood with each beat, we should expect to find that the cubic content of the normal auricle was somewhere about half of this amount.

But, as the normal capacity of the auricle is at least 3 ounces, there must be some error in this supposition. The error lies, I believe, in supposing that there is any considerable general emptying of the auricle during the auricular systole.

---

[1] Essay XII, p. 429.

It is probable, as was suggested by Mayo Collier,[1] in 1889, that the function of the auricle is only to throw a small quantity of blood into a nearly full ventricle, by means of the highly muscular appendix, for the purpose of closing and stretching the auriculo-ventricular valves prior to the commencement of systole; and that the body of the auricle has little, if any, power of emptying itself by its own contraction. This function, therefore, of the body of the auricle is to act as a reservoir, rather than as an accessory force-pump for filling the ventricle, and the function of its muscular fibres is simply to make its walls tense enough for the action of the appendix to take place (*vide* p. 529).

This view of the function of the auricles fits in very well with the demands of the theory which we are discussing.

If the auricle does not empty itself to any extent at the time of the auricular systole, and is only emptied by the aspiration of the ventricle, it follows that that aspiration must draw into the ventricle the greater part of its full charge of blood—namely, nearly 3 ounces. [If this is so, it is possible that, under certain circumstances, the reception of this full charge may cause a preliminary closure of the tricuspid or mitral valve by its tendency to escape back into the auricle, and that the faint sound, which is sometimes heard at the commencement of the phase of relaxation, and which I call the third sound of the heart, may sometimes be caused by this quiet closure of these valves.[2]]

We may, I think, assume that the body of the auricle acts simply as a reservoir, in which blood collects, and thus enables what is practically a full charge of 3 ounces to be drawn into the ventricle during its expansion phase.

CLINICAL EVIDENCE AS TO THE NATURE OF THE EXPANSION MOVEMENT

It is not easy to obtain direct clinical evidence as to the nature of the expansion movement, but such evidence as is obtainable is all in favour of this theory, for by it all the phenomena of valvular disease can be explained very much more satisfactorily than by the older theory.

---

[1] *The Physiology of the Vascular System*, p. 10.

[2] It is more probable that the tonic contraction of the auricle and great veins suffices to keep the blood in the relaxed ventricle.

As to mitral regurgitation and stenosis :

According to the accepted theory, when compensation fails in mitral disease, extra work is thrown upon the right ventricle by the delay in the pulmonary circulation, and the re-establishment *and maintenance* of compensation depend upon the success, or otherwise, of the efforts of the right ventricle to overcome this.

When the power of the ventricular expansion is realised, this statement must be modified, so far as the *maintenance* of compensation is concerned, for this must depend upon the success of the left ventricle in compensating for its own defects. If the left ventricle fails, then, and then only, is extra work thrown upon the right ventricle, and its assistance called for.

MITRAL REGURGITATION

Adequate compensation for mitral regurgitation must consist in both dilatation of the ventricular cavity, and hypertrophy of its walls (see Davies's 'Circulation through Diseased Hearts,' 1887), i.e. *dilatation* enough to enable the heart to hold the normal charge, *plus* the amount which will regurgitate into the auricle and *hypertrophy*, so that it may have strength to throw this extra amount.

Now, according to the theory under discussion, this change in the heart will not only suffice, so far as the systolic work of the ventricle is concerned, but will also give the extra power necessary properly to fill the ventricle during diastole. For this increase, both in its cubic content and strength, will increase its capability of aspirating blood from the auricle, and to the extent rendered necessary by the amount of the leakage. Therefore, at the end of the expansion movement in compensated mitral regurgitation, the auricle will be as fully emptied as normal, and the pulmonary circulation and the right side of the heart will not be hampered.

In proof of this, I have measured the loudness of the pulmonary second sound in cases of compensated mitral regurgitation, and I find that the true loudness of this sound does not exceed the normal limits, thus pointing to the probability that the right ventricle, in such cases, is not ordinarily called on to do more work than normal.[1]

There is usually some apparent accentuation of this sound in these cases, but this is due to the sound being simply sharper in tone, probably owing to the right ventricle increasing the force of its beat, in harmony with the action of the left, and it does not suggest any excess of pressure in the pulmonary artery as will be explained more fully later on, under mitral stenosis.

MITRAL STENOSIS

It is from mitral stenosis that the expansion theory receives the most clinical support, for it here sweeps away the difficulties which used to surround the explanations of the clinical phenomena associated with this condition.

PROCESS OF COMPENSATION

In order to overcome the hindrance to the entrance of blood into the ventricle, greater aspiratory force is needed. This, according to the theory we are discussing, could be attained by the ventricle altering its beat.

In order to develop the maximal amount of suction during the short expansion period, the force of the ventricular contraction would have to be concentrated upon the very end of the systole so as to call forth the greatest possible amount of elastic and muscular recoil from the ventricular wall. But this change in the character of the beat *is* observable in mitral stenosis, for the first sound is abnormally loud, and also it ends very sharply, just as might be expected if it were trying to increase its suction power. I have heard the first sound of the heart 2 feet away from the patient, in a case of mitral stenosis, whereas normally it cannot be heard with a binaural stethoscope ⅛ inch away from the chest wall. Further evidence on this point is given by the fact that, where the heart in mitral stenosis is temporarily embarrassed, the loudness of the first sound is often very noticeably increased for a time, and far in excess of the increase noticed in similar circumstances in health, or in other forms of heart failure. In such a case, the first sound returns to its usual loudness as the heart recovers itself.[1]

---

[1] Essay XI, p. 423.

This change in the character of the first sound is, therefore, evidence in favour of the expansion theory, though it does not give special support to the theory of muscular, as apart from elastic, expansion.

Again, the fact that compensation depends upon the muscular action of the left ventricle accounts for the hypertrophy which is often noticeable in mitral stenosis, and which, according to the older theory, would not be so easily explained; also, in cases of death in mitral stenosis without rupture of compensation, it has been noticed that the left auricle is of normal size, and not dilated. This is what would be expected, if compensation were wholly due to the increased suction power of the ventricle, but would be very hard to account for by the older theory of compensation.

PULMONARY SECOND SOUND IN MITRAL STENOSIS

This theory of the freedom of the auricle from any embarrassment, in cases of well-compensated mitral stenosis, is further emphasised by the character of the pulmonary second sound, for, in the majority of these cases, which I have carefully measured, it does not exceed the normal limit of loudness, and in some cases is well within that limit. This statement may appear to conflict with what is a well-recognised sign of mitral stenosis—namely, accentuation of the pulmonary second sound, but it does not really do so. In most of these cases, the second sound, when heard by the stethoscope, *appears* to be much louder than normal; but, if some means are taken of deadening the sound, it will be found that its penetrative power is not increased, showing that there is no increase in the volume of the sound, as would be the case if it were due to increased tension in the pulmonary artery. This can be easily demonstrated by inserting a deflated rubber toy balloon under the stethoscope, and slowly lifting it by inflating the balloon through a tube with the mouth. So long as the stethoscope is within ½ or ¾ of an inch of the chest wall, the sound retains its loudness; but, when the stethoscope is raised higher, the sound is rapidly damped down, and is extinguished at 1 inch or 1⅛ inches, as is the case with a normal second sound. In uncompensated mitral stenosis, on the other hand, where the right ventricle *is* doing extra work, the sound may often be heard as far away as 1⅜ or 1½ inches or more.

---

[1] Essay XI. p. 425.

In other conditions, where the right side of the heart is doing extra work, true increase in the loudness of the pulmonary second sound may also be observed. For instance, in some cases of anæmia, in which the tension in the pulmonary artery is certainly increased, the second sound may be heard when the stethoscope is raised as much as 2½ inches from the chest wall instead of the normal 1⅛ inches, and, in such a case, the sound may even be audible, without the interposition of the rubber bag, with the stethoscope ⅛ or ¼ inch from the chest wall. Such a degree of accentuation as this I have never seen in fully compensated mitral stenosis or regurgitation, although it surely ought to occur if the maintenance of compensation really depended upon the work of the right heart. I have not infrequently noted an accentuation nearly as great as this occurring during the process of compensation, but which materially lessened as compensation became more perfect.

Spurious Accentuation of the Second Sound

The explanation of the apparent loudness of the pulmonary second sound, in these cases of compensated mitral disease, is to be found in the fact that the right ventricle cannot but partake in the forcible action of the left ventricle, and the increase of suction, which results therefrom, causes the blood in the pulmonary artery to return with greater rapidity against the pulmonary valves; but, as there is only the normal volume of blood in the artery, although the *sharpness* of the sound may be increased, its *volume* is not. It is the difference between the sound of a sharp blow with a small hammer and the more penetrating sound of a less sharp blow with a heavier hammer.

This absence of a true accentuation of the pulmonary second sound in mitral stenosis is therefore evidence that the increased ventricular aspiration is sufficient effectively to meet the altered conditions, and prevent extra work from being thrown upon the right ventricle when compensation is complete. The healthiness of the lungs, and the freedom from all signs of pulmonary congestion, which can often be seen in compensated mitral disease, is further evidence in support of this view.

MID-DIASTOLIC MITRAL MURMUR

This theory of active ventricular expansion also suffices to explain the mid-diastolic murmur of mitral stenosis, much more effectively than the other theory.

The loudness of this murmur, and the presence, as can often be noted, of a well-marked thrill with it, points to its being due to some such force as that of ventricular aspiration, as was first pointed out by Dr. Lockhart Gibson, then of Edinburgh, in the *Lancet* for 1884; as was also the fact that this murmur often increases in loudness, as compensation becomes more effective.

The muscular nature of this expansion movement also affords an explanation of the reason why this murmur does not occur, as it might reasonably be expected to do, at the time of the closure of the semilunar valves, when the blood is, presumably, beginning to enter the ventricle.

This explanation will probably be found to be somewhat as follows :—

During the expansion phase, the cardiac muscles, as already stated, are still in a state of activity, albeit elongating instead of contracting, and the musculi papillares may reasonably be expected to partake in that activity. This being so, it is quite possible that, during the whole of this expansion phase of the cardiac cycle, the chordæ tendineæ are kept tense by them, and although the aspirated blood is flowing freely through the valve, its segments (being tense) are not free to vibrate in the blood stream.

When, however, relaxation comes on suddenly at the end of this phase, they *are* free to vibrate in the blood stream which continues to flow through the valve under the momentum imparted to it during the brief expansion phase.

According to this theory, the fixation of the valve segments, by the activity of the musculi papillares, prevents the first inrush of the blood from causing an early diastolic murmur in mitral stenosis, and delays its commencement till the onset of relaxation, which takes place, as before said, one-tenth of a second after the closure of the semilunar valves. To this delay, the murmur owes its name of mid-diastolic (or, as it used to be called, post-diastolic) mitral murmur.

If the second sound and the commencement of the mid-diastolic murmur are recorded by an electric signal upon a revolving drum, it will be found that the interval between them is about a tenth of a second; or, if the murmur is recorded on the cardiogram, it will be found to occur at the point where the heart muscle relaxes.

MID-DIASTOLIC MURMUR IN AORTIC REGURGITATION

Further confirmation of this theory, as to the mode of production of the mid-diastolic murmur, is furnished by a murmur which may not at all infrequently be heard in aortic regurgitation. I refer to a so-called presystolic, which may be mistaken for a true Austin Flint murmur.

If this murmur is carefully timed, it will be found that it follows the second sound by the same interval as does the mitral mid-diastolic—namely, a tenth of a second; but, as in aortic regurgitation the heart is often rapid, and the diastole short, this interval of a tenth of a second brings it so near to the following first sound of the heart that it appears to be presystolic. Nevertheless, careful timing will show that its relationships are with the second sound and not with the first, for its relationship to the first sound alters, when the heart alters its rate, whereas its relationship to the second sound remains constant. It is, therefore, mid-diastolic and not presystolic.

In order to understand this murmur, it must be remembered that the anterior cusp of the mitral valve forms the posterior wall of the channel down which the regurgitant blood flows in aortic regurgitation.

During the expansion phase, while the valve segments are, according to supposition, held tense, they do not vibrate; but if the regurgitation is sufficiently prolonged and copious, it will cause vibration of the anterior segment of the mitral valve at the onset of relaxation. Careful mapping out of the area, over which this murmur is best heard, helps to confirm this theory; for it is often best heard over the body of the right ventricle, internal to the apex, over an area, where the vibrations of the anterior cusp of the mitral valve might well be conducted to the anterior wall of the chest, by the blood stream, which separates it from the muscular tissue of the septum, and of the right ventricle. The ordinary mid-diastolic murmur of mitral stenosis is not, as a rule, heard over this area so clearly as is the Austin Flint murmur, because the channel to the aorta is not, in this case, distended with blood, and there are not, therefore, the same facilities for conduction to the anterior wall of the chest.

In some cases, the mid-diastolic aortic regurgitant murmur has only the same area of audition as the mitral mid-diastolic —namely, at and internal to the heart's apex.

This explanation is further upheld by the observation that sometimes this spurious Austin Flint murmur is quite faint, and is simply a mid-diastolic accentuation of the ordinary regurgitant murmur audible at the sternal end of the fourth or fifth left interspace, thus showing that it is not a separate murmur, produced elsewhere, but is due to the vibration of the same stream of blood, which is producing the aortic diastolic murmur.

THE THIRD SOUND OF THE HEART[1]

In cases in which the flow of the blood is not strong enough to make the valve segments actually vibrate, the onset of relaxation is in mitral stenosis marked by a short sound, which is heard at the apex, and may be nearly as loud as the second sound itself, but follows it at the same interval (a tenth of a second) as the murmur would do.

---

[1] See Essay XII.

This sound, which is usually called a 'spurious,' or 'apical' reduplication of the second sound, is well known as occurring in those cases of stenosis which show a true presystolic murmur.

This third sound marks the commencement of the third, or relaxation, phase of the cardiac cycle, just as the second sound marks that of the second, or expansion, phase.

It may now be asked why this third sound does not occur in the normal heart if this theory is correct, and the answer is, that, in the normal heart, the rapid flow of blood through the mitral valve has ceased by the time relaxation sets in, and such blood as may then be passing through it is unable to move the valves suddenly enough to produce a sound. The reason for this becomes apparent, if the size of the mitral orifice is contrasted with the capacity of the auricle. In the normal heart, the opening is wide enough to allow the whole of the 3-ounce charge to pass into the ventricle during the expansion phase, in spite of its only lasting so short a time; for the diameter of the full auricle would be about 2·15 inches, and the diameter of the mitral orifice is 1·18 inches.

According to this supposition, anything which delays the emptying of the auricle, and so leads to the prolongation of the main flow of blood from it till the relaxation sets in, ought to produce this third sound of which we are speaking, whether it is narrowing of the mitral orifice, as in mitral stenosis, or an excessive amount of blood in the auricle, or a defective aspiratory power on the part of the ventricle.

The correctness of this theory is indicated by the fact that we find this third sound, clinically, in just such cases as we have indicated.

We not infrequently find a third sound present over the right ventricle in anæmia, due to either or to both of the last two causes we have just enumerated, for the ventricle is often acting weakly in such cases, and the auricle is over-distended with blood.

A third sound may also possibly be produced in the manner referred to earlier—namely, by the mitral valve being closed immediately after the expansion phase, by the endeavour of the aspirated blood to return from the ventricle back into the auricle (*vide* p. 388).

This early closing of the mitral valve may be the cause of the third sound of the heart in some cases of mitral disease, and it is also possible that it may explain the occurrence of a third sound over the right ventricle in certain cases of anæmia with dilatation of the right auricle.

COMPENSATORY DILATATION OF THE LEFT VENTRICLE

There is one other point, where this theory of active expansion solves what would otherwise be a problem not easy of solution, for it explains the compensatory dilatation of the ventricle in mitral regurgitation.

It is evident, where the mitral valve is incompetent, that at the end of the systole, the auricle must contain, in addition to its normal charge of 3 ounces, the amount which has leaked back from the ventricle. This additional amount will, therefore, be aspirated into the ventricle, during its expansion phase, and will cause its dilatation. The ventricle is dilated by means of its own aspiratory force. Evidence of this is given by cardiographic records, in cases of mitral regurgitation, for a sharp-pointed rise in the cardiogram is often observable after the commencement of the relaxation period, due to the impact of the aspirated blood upon the relaxed ventricular wall.[1]

It is not always very safe to arrive at conclusions by a process of exclusion; but, in the present instance, it is justifiable to conclude that a force, which can distinctly raise the lever of a cardiograph applied over the left ventricle, after the ventricular wall has relaxed (i.e. after the ventricular and before the auricular systole), must be due to the impact against it of the blood which has been set in motion by the ventricular aspiration.

The mechanism responsible for this distension of the ventricle during its relaxation period is best illustrated by a well-marked case, such as that of a girl who died in the General Hospital from mitral regurgitation, and with adherent pericardium. The adhesion having been prior to the onset of the regurgitation, the rigidity of the ventricular wall, thus brought about, prevented compensatory dilatation of the left ventricle from taking place. The result was that, at the post-mortem, the left ventricle was practically of normal size, holding only 90 c.c., whereas the left auricle held 164 c.c. In such a case as this, when the left ventricle expanded, it would, by its suction, set the 164 c.c. in motion, and the momentum of the blood would tend to force more than the 90 c.c. into the ventricle. The elasticity of the ventricle, strengthened as it was by the adherent pericardium, would expel the balance over and above the 90 c.c. back into the auricle through the leaky mitral valve. The backward reflux, following the transient over-distension, would give rise to just such a sharp-pointed wave as the cardiographic tracing showed.[1]

---

[1] Essay X, p. 407, and Essay XII, p. 433.

The case, therefore, shows us that the momentum of the blood, aspirated into the ventricle during its expansion phase, is very considerable, and is able to over-distend the left ventricle when its walls are relaxed.

It also seems reasonable to assume that, had it not been for the pericardial adhesions, this distensile force would have been amply sufficient to cause dilatation of the ventricular cavity, until its size approximated to that of the auricle: in other words, to produce the necessary *compensatory dilatation* of the ventricle.

In this case, although there was no mitral stenosis, a loud third sound was audible over the heart, as would be expected according to the theory of its causation, as already stated.

Where the right ventricle is constricted in a similar way, by adherent pericardium, a similar reflux of blood from the undilatable ventricle gives rise to a distinct diastolic impulse in the veins of the neck, and the presence of this wave, which is clearly visible, is a sign of ill-omen for the patient.

These, then, are the points in favour of the theory of cardiac action which I am advocating, and they appear to me, when taken together, to give undoubted support to the view that true muscular expansion plays a most important part in the heart's action.

---

[1] Essay X, p. 407, and Essay XII, p. 433.

If this is so, it would be well to alter the nomenclature of some of the heart's sounds and murmurs as follows :—

The *first* sound marks the commencement of ventricular contraction or systole.

(Contraction or systolic murmurs may be produced at mitral, aortic, tricuspid valves, &c.)

The *second* sound marks the commencement of expansion.

(An expansion murmur occurs in aortic regurgitation.)

The *third* sound (when present) marks the onset of relaxation.

A relaxation murmur may occur :—

1. In mitral stenosis, &c.
2. Occasionally, at the tricuspid orifice in dilatation of the right heart.
3. As an addition to the expansion murmur in aortic regurgitation.

ESSAY X.—THE DIASTOLIC EXPANSION MOVEMENT OF THE VENTRICLES AS A FACTOR IN COMPENSATION FOR DISEASE OF THE MITRAL VALVE [1]

MY object in this paper is to bring forward evidence in favour of the heart having the power of enlarging its chambers by means of a true muscular expansion movement. The possession of this power would constitute the heart a double-acting pump instead of its having the power of single action only with which accepted theories credit it. It would then be able to fill itself by its own counterstroke, just as is the case with most of the pumps which the art of man has devised. The possession of such a power would mean that the heart is a much more perfect mechanism than if it had to depend upon some extraneous mechanism for the filling of its ventricles, as the ordinarily accepted theory asserts.

This view—that the ventricles of the heart possess the power of aspiration in virtue of a true vital expansion of its muscular fibres—was, I believe, first definitely propounded by Dr. Lockhart Gibson, when assistant to the late Professor Rutherford, some sixteen years ago.[2] He pointed out that in the case of ordinary striated muscular fibre the phase of contraction, which results from a minimal stimulation of the muscle, is followed by a phase of sudden expansion, during which the muscle develops about two-fifths of the force it develops during contraction, but in, of course, the reverse direction. The possession of such an expansile power by the individual muscular fibres of the heart would cause a sudden and moderately powerful enlargement of the ventricles at the commencement of the disastole.

---

[1] In part taken from a thesis (Gold Medal) for the degree of M.D. at the University of Edinburgh, 1892. *Vide* the *British Medical Journal*, September 29, 1900.

[2] *Lancet*, April 19, 1884, p. 730.

PHYSIOLOGICAL EVIDENCE AS TO THE NATURE OF THE EXPANSION MOVEMENT OF THE VENTRICLES

So far as I have been able to ascertain, there is but little experimental evidence that the expansion of the ventricles is due to a muscular movement in addition to simple elastic resiliency. We know, however, that the muscular fibre of the ventricles remains active for an appreciable time after the closure of the semilunar valves, and does not at once enter upon its phase of relaxation. The most striking physiological evidence of the possession of such a power which I have been able to find is afforded by some experiments made by Stephani and quoted by Tigerstedt.[1] He found that section of the vagus nerve diminished the force of the heart's recoil after contraction. When the vagus was divided, less force was required to prevent the diastolic enlargement of the heart than was necessary before section of the vagus.

The confirmation of this observation would seem to prove that the expansion of the heart is, in part at all events, a muscular movement under nervous control, and not simply a mechanical phenomenon due to elasticity only.

CLINICAL EVIDENCE OF THE NATURE OF THE EXPANSION MOVEMENT IN THE NORMAL HEART

Clinically, we have little opportunity of studying the changes in volume which the heart undergoes during its phases of contraction and expansion. Were instantaneous radiography of the heart possible, we should have an excellent means.

Our chief source of information is the cardiograph. There are difficulties and fallacies connected with the use of the cardiograph of which we must take account. Of these the interpretation of the cardiogram is perhaps the most important. This I have in large measure overcome by the simultaneous record of the cardiac sounds upon the cardiogram. This can be done by means of an electric time-marker, which makes an audible click as it rises and falls. It is quite easy in most cases to synchronise the clicks of the electric signal with the heart sounds, and thus to distinguish between the systolic and the diastolic portion of the cardiogram.

---

[1] *Phys. des Kreislaufes*, p. 143.

In the course of the last nine years, since I first used this method, I have had ample evidence of its reliability; and, further, I think I can claim that the beautiful researches of M. A. Chauveau, upon the movement of the cardiac valves in relation to the intracardial pressure curves and the cardiogram,[1] fully confirm the interpretation of the cardiograms which I am about to give.

In using the cardiograph for the study of the expansion movement of the heart, we must remember the limited area over which we can take tracings, and give due allowance for the changes in form which may occur apart from alterations in the volume of the heart.

EVIDENCE OBTAINED FROM THE STUDY OF THE HEART IN VALVULAR DISEASE

When considering the process of compensation for incompetence or stenosis of the mitral valve, the possession of such a muscular power as we are discussing would be of very great service to the heart. In the case of mitral incompetence the occurrence of hypertrophy and dilatation would increase the aspiratory power equally with the expulsive power. If this were so, we would expect in a case of compensated mitral regurgitation that there would be little, if any, backward pressure on the lungs or right side of the heart, and that the left auricle would have no more than a normal amount of work to do.

PATHOLOGICAL EVIDENCE

In the only cases of mitral regurgitation with unbroken compensation which I have been able to examine post-mortem there was evidently much regurgitation, and the left ventricle was much dilated, but the left auricle was of almost normal size, suggesting that the hypertrophy and dilatation called forth by the failure in expelling power was fully sufficient for compensation so far as aspiration also was concerned.

---

[1] *Jour. de physiol. pathol.* 1899–1900.

In the first of the cases the left ventricle held 165 c.c. as against the normal 80 c.c., while the left auricle held the normal 90 c.c. In the other case the left ventricle held 235 c.c. as against the normal 80 c.c.; the left auricle only held 115 c.c. Considering that it is quite usual for the left auricle to be dilated to the extent of 120 c.c. in patients dying of pneumonia and other diseases without definite cardiac disease, these figures are remarkable, and cannot, so far as I can see, be explained by the ordinarily accepted theory of compensation in mitral regurgitation. They are, I consider, strong evidence in favour of what I may call the '*vis a fronte*' theory of compensation—namely, that the ventricle by increase in its aspiratory power compensates for the incompetence of the mitral valve, and that so long as compensation remains unbroken the left auricle and right side of the heart have very little extra strain put upon them.

In comparison with the above figures those of another case (Case IV) are striking. Here compensation had failed, and while the left ventricle only held 200 c.c., as compared with the 235 c.c. above mentioned, the left auricle held 250 c.c. as against the 115 c.c. where compensation was good. In such a case there was undoubtedly extra strain on the left auricle and right ventricle.

Cardiographic Evidence in Mitral Regurgitation

In mitral regurgitation, we have cardiographic evidence of increased diastolic expansion of the ventricles. I show some tracings taken from cases of mitral incompetence, and side by side with them some from normal hearts. The time of occurrence of the heart sounds is marked on some of them. In tracings taken over the apex beat, the tilt of the heart's apex hides any movement due to change in volume, because, as has long been known, the apex remains pressed against the chest wall for an appreciable time after the closure of the semilunar valves. In tracings taken over the ventricle internal to the apex, the diminution in the size of the ventricle during systole causes the heart to recede from the chest wall, and we have more or less of a systolic depression in the cardiogram (*vide* Plate VII, Case I, figs. 3 and 4). In such a situation the enlargement of the heart, after the systole, may also be expected to show as a rise in the cardiogram if sufficiently powerful. In taking my tracings, I used a pressure of about ¼ lb. in the spring of my cardiograph, and therefore any elevation seen in the tracings I show implies that the heart wall was able to overcome a resistance of at least ¼ lb. in causing the elevation of the lever.

I show some tracings from a normal heart where there is some evidence of sudden enlargement at the time of the closure of the semilunar valves (Plate VII, Normal Heart, fig. 2).

I also show some tracings taken over the right ventricle in a case of anæmic dilatation. They show very well the sudden enlargement of the ventricle after the systole (Plate VII, Case I, figs. 2, 3, and 4). The fact of the lever of the cardiograph rising so rapidly and powerfully is proof that the ventricular wall does not relax till after a certain amount of expansion has occurred, otherwise it would not have strength to lift the lever. I may say that this is quite an ordinary type of tracing. In comparison with these, I show some tracings from a case of moderate mitral regurgitation. In this series the diastolic expansion wave is well marked (Case II).

In the next two series (Cases III and IV) there was very considerable regurgitation. In the first the systolic diminution in size of the ventricle is much more rapid than normal, as might be expected, and the diastolic expansion rise is proportionately more marked. In the next (Case IV), there was very extreme mitral regurgitation, the cavity of the left ventricle was enlarged to more than twice its natural size, its contents being 7 ounces (200 c.c.) instead of the normal 2⅞ ounces (80 c.c.); the contents of the left auricle were 9 ounces (250 c.c.). In this case the systolic diminution was so extreme and the diastolic expansion so great that the tracing is divided into two nearly equal halves by a deep notch.

Case V.—Finally, I show you some tracings taken over the right ventricle in a case of mitral regurgitation with extreme secondary tricuspid regurgitation. So extreme, indeed, was the regurgitation that a systolic thrill could be felt over the right auricle in the region of the right nipple. The tracings are extremely interesting but too complicated to be fully explained within the limits of this essay. I show you, however, a series from the outer part of the fifth left interspace.

I have proved the correctness of the interpretations given in the figures by means of many observations with the electric recorder of the heart sounds, and by the polygraph, and also by taking consecutive series of tracings ⅛-inch apart over the available part of the heart. The diastolic expansion wave is here very high, and in tracings taken in the fifth interspace in the nipple line—the apex being in the axilla—this part of the tracing is far higher than the rest (*vide* Plate XII).

In fig. 5 is shown a polygraph tracing taken with a Marey's drum cardiograph, with the heart sounds recorded on it. It shows that the closure of the semilunar valves occurs at the notch about half-way up the diastolic expansion rise. It is difficult for me to believe that these phenomena can be explained on the supposition of the expansion movement being simply a mechanical one due to the elastic recoil of the relaxing heart wall. There is far more and far stronger movement than could be explained except by a true muscular expansion movement.

Clinical Evidence in Mitral Stenosis

In mitral stenosis, we have clinically various phenomena more or less indicative of increased diastolic activity on the part of the ventricles. In the first place, there is sometimes post-mortem evidence that the left ventricle has increased work to do. Its wall sometimes seems to be slightly thicker than normal. Secondly, the first sound of the heart is louder than normal in mitral stenosis. Now the ventricle has, if anything, a diminished amount of work to do so far as its direct or forward work is concerned, owing to the hindrance to the filling of the ventricle, and we ought therefore to expect that the first sound would not be louder than normal. Thirdly, the first sound ends more sharply than normal.

The explanation of these facts is, I believe, that the ventricle alters its beat. It contracts with greater force and suddenness, in order that the recoil—both elastic and muscular—may be as great as possible, thus developing the maximum amount of aspiration. In this way the ventricle by its altered beat is able to compensate for the valvular disability, without there being any permanent need for increased action of the right heart. Every practitioner must know many cases of mitral stenosis which are fully compensated, and where there is no evidence at all of any increased blood pressure in the lungs. According to the ordinary accepted *vis a tergo* theory of compensation, there must be increased pulmonary blood pressure in all cases of stenosis. According to the *vis a fronte* theory, this is not necessary, and the clinical facts seem to me more in favour of the latter than the former.

I shall deal with the other clinical evidence of increased aspiratory action after dealing with the cardiographic evidence.

Cardiographic Evidence in Mitral Stenosis

Owing to the ventricle filling more slowly than normal, we expect the expansion wave to be less marked than normal, and we shall not expect it to show in the cardiographic tracings. This is so (*vide* Case VI, figs. 1, 2, and 3). Although the expansion movement itself does not show in the tracing, we very often have an accessory wave which gives us very important evidence as to the presence of a powerful aspiratory action. I refer to a small sharp wave which occurs just after the diastolic expansion movement has ceased—namely, immediately after relaxation of the cardiac muscle has occurred. This wave is best seen at and near the apex (*vide* Cases VII and VIII), but is sometimes evident over a considerable part of the heart. The cause of this wave is, I believe, as follows: At the commencement of diastole the left auricle is naturally considerably distended with blood, and the ventricular aspiration causes it to rush into the ventricle with considerable force. Owing to the narrowness of the mitral orifice it enters more slowly than normal, and there is not time for all the blood to get through into the ventricle before the expansion movement ceases. When, therefore, relaxation commences there is still a strong stream of blood flowing into the ventricle. It is the impact of this stream upon the relaxed heart wall that causes the small rise referred to. (This wave is clearly not due to the auricle, because when the heart is irregular this wave maintains a constant relation to the beat which precedes it, while its distance from the following beat is variable (*vide* fig. 142, p. 432).

SUCTION RECOIL WAVE

If this theory be correct, we ought to see this wave (which I call the suction recoil wave) whenever the auricle is not able to discharge the bulk of its contents into the ventricle before the onset of relaxation. I show a tracing (Case VII) from a case where there was considerable mitral regurgitation with no stenosis, but where the ventricle could not dilate to its proper amount in consequence of pericardial adhesions. The cubic content of the left ventricle was 90 c.c., and that of the left auricle 164 c.c. In this case the suction recoil wave was most marked (*vide* p. 433).

This suction recoil wave is closely associated with a diastolic cardiac sound which is, I believe, due, like it, to the aspiratory power of the ventricle. I refer to the so-called reduplicated second sound which is heard at the apex in mitral stenosis. This sound has no right to be called a second sound. It can easily be demonstrated that it is neither the aortic nor the pulmonary second sound. With a differential stethoscope it is frequently easy to hear all three sounds. This sound ought to have a name of its own, and I have named it the 'third sound' of the heart, and called it so for many years. It has been described by some authors as the sound of the mitral opening. This is, I believe, not quite correct, for it is produced shortly after the opening of the mitral valve. I have in many instances recorded the time of occurrence of this sound on the cardiogram, and I find that it usually coincides exactly with the commencement of the suction recoil wave just spoken of. I believe that this sound is caused by the inrush of blood into the ventricle at the commencement of diastole, as was suggested by Dr. Sansom, twenty years ago,[1] but the exact mechanism of its production I believe to be as follows :—

During the phase of diastolic expansion, the auriculo-ventricular valve is kept more or less rigid by the tension of the chordæ tendineæ. As soon, however, as relaxation sets in, the valve is free to move, and if the auricle has not already been emptied by the aspiration during the expansion movement, the in-rushing blood stream will flap the valve back against the ventricle wall in a manner calculated to produce the faint sound we are speaking of.

---

[1] *Proc. of Med. Soc. of London*, vol. v. p. 199 *et seq.*

MITRAL MID-DIASTOLIC MURMUR

I now come to the last of the physical signs in mitral stenosis of which I wish to speak—namely, the mitral diastolic murmur. By this murmur, I mean the one sometimes called post-diastolic, because of its occurring after the second sound, not with it as in the case of the aortic diastolic murmur. This murmur is also sometimes, I believe, called a mid-diastolic murmur. It begins loudly and then fades away, although sometimes prolonged through the diastole. Its time relation to the second sound is constant, but its relation to the first is not constant, the reverse being true of the presystolic. This murmur is now, I believe, very generally recognised as being a suction murmur and due to ventricular aspiration, and the fact that it is very often strong enough to give rise to a palpable thrill is evidence as to the force of the aspiration.

On studying the relation of this murmur to the 'third sound' of the heart it is evident that the two are closely related. The murmur occurs at the same interval of time, after the second sound, that the third sound does; and in cases where the third sound is audible, a slight alteration in the rate or strength of the heart will frequently transform the third sound into the mid-diastolic murmur and *vice versa.* The mid-diastolic murmur may accompany or replace the third sound. Also on recording the time of this murmur on the cardiogram it is found to coincide with the suction recoil wave just as the third sound does. The explanation given for the third cardiac sound holds true, I think, for this murmur. The valve segments are not free to vibrate in the blood stream till after the muscular relaxation has set in.

Cardiogram in Aortic Regurgitation

The last point to which I wish to draw attention in this essay is the evidence given by the cardiogram in aortic regurgitation. If the diastolic expansion movement were purely a mechanical one it ought to be evident in aortic regurgitation as it is in mitral regurgitation. The cardiogram shows that this is not so. A marked feature of all the tracings I have taken in aortic regurgitation is the absence of the diastolic expansion wave. The tracings shown under Case IX, fig. 1, and Case X illustrate this point, and show the absence of the expansion wave.

In giving these tracings as typical of the cardiogram in aortic regurgitation, I ought to add that my generalisation as to aortic disease is only based upon the cardiographic study of some eight or ten cases. There was, however, sufficient similarity in the tracings obtained to make it admissible to generalise from so few a number. The reason for this type of tracing is evident, for in aortic regurgitation increase of ventricular aspiration would only increase the amount of leakage. The heart, therefore, alters its beat so as to produce a minimal amount of diastolic expansion.

The tracings in aortic regurgitation, therefore, give us most important evidence that the expansion movement is one under nervous control and not a simple mechanical one.

In bringing this essay to a conclusion, I am conscious that much of it is theory, and therefore may be erroneous. But as a result of many years' careful observation and research, I have been unable to obtain any evidence except such as seems to point strongly in favour of the expansion of the ventricles being in large measure due to a true muscular movement.

EXPLANATION OF PLATES VII AND VIII

The tracings are all to be read from left to right.

They were all taken with a Galabin's cardiograph, except Case V (fig. 5) and Case VI (fig. 3). These two were taken with a Marey's drum cardiograph and tambours.

The systolic portion of the cardiogram is enclosed between broken lines, and is marked by a black band at the foot of each tracing. The position of the line marking the close of the ventricular systole has in most of the instances here given been ascertained by recording on the cardiogram the time of occurrence of the second sound. In the remainder it has been inferred, owing to the similarity of the tracings to others in which the time of occurrence of the second sound had been recorded. This line marks the commencement of the period of rapid diastolic expansion of the ventricles. The close of this expansion period (i.e. the commencement of complete ventricular relaxation) is marked by a dotted line. At the foot of each figure the portion of the cycle occupied by the expansion period is marked by a dotted band.

PLATE VII

CASE I.—ANÆMIA WITH DILATATION OF THE HEART.—The apex was in the fourth interspace, 3¼ inches out from the sternum. Figs. 1, 2, 3, and 4 are tracings taken in the fourth interspace at distances of 3¼ inches, 3 inches, 2¾ inches, and 2¼ inches respectively from the sternum. They show the gradual substitution of a systolic depression for the systolic elevation shown in the apex tracing. This depression is due to the withdrawal of the heart from the chest wall, owing to its systolic diminution in volume. These latter tracings show a well-marked expansion rise at the commencement of diastole.

CASE II.—MITRAL REGURGITATION WITH ANÆMIA.—Fig. 1, tracing from the apex in the sixth interspace, 3 inches out from the sternum (i.e. ½ inch external to the nipple line). Figs. 2, 3, 4, and 5 are tracings from the fifth space, at distances from the sternum of 3 inches, 2¾ inches, 2½ inches, and about 2¼ inches respectively.

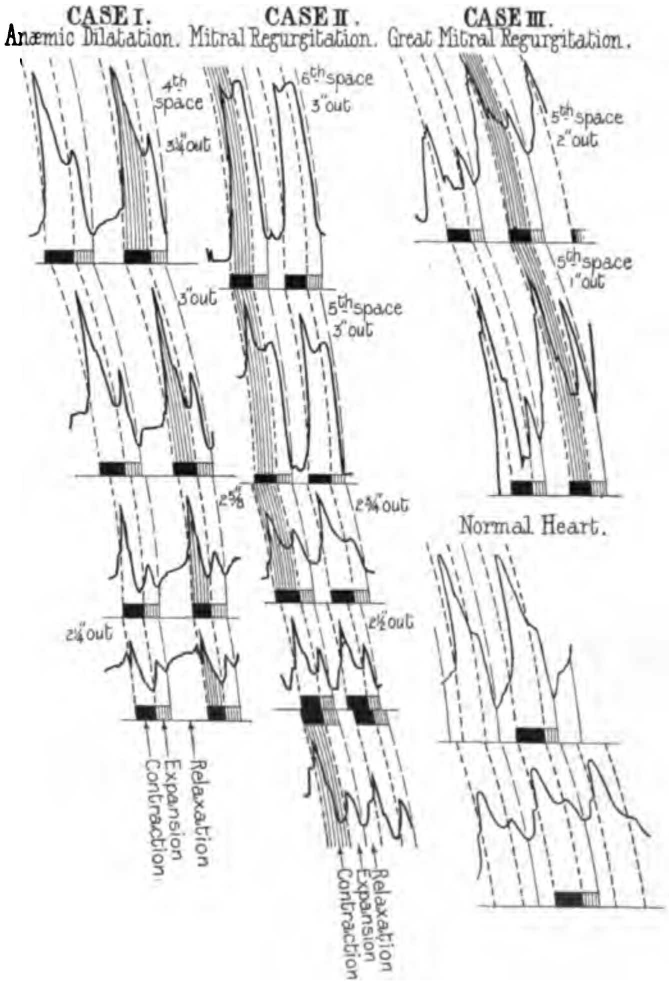
They show, as in Case I, the substitution of a systolic depression for the systolic elevation of the apex cardiogram, and they show a well-marked diastolic expansion rise.

CASE III.—CONSIDERABLE MITRAL REGURGITATION.—The apex was in the sixth space, 2 inches external to the nipple line. The apex cardiogram was similar to Case II, and is not given. Fig. 1 and fig. 2, tracings from the fifth space, 2 inches and 1 inch out from the sternum. Both tracings show the expansion movement well, but especially fig. 2.

Normal heart, from a case of fibroid phthisis, with retraction of the left lung. Fig. 1 and fig. 2, cardiograms taken at the apex just internal to it. The latter shows a slight amount of diastolic expansion.

PLATE VII

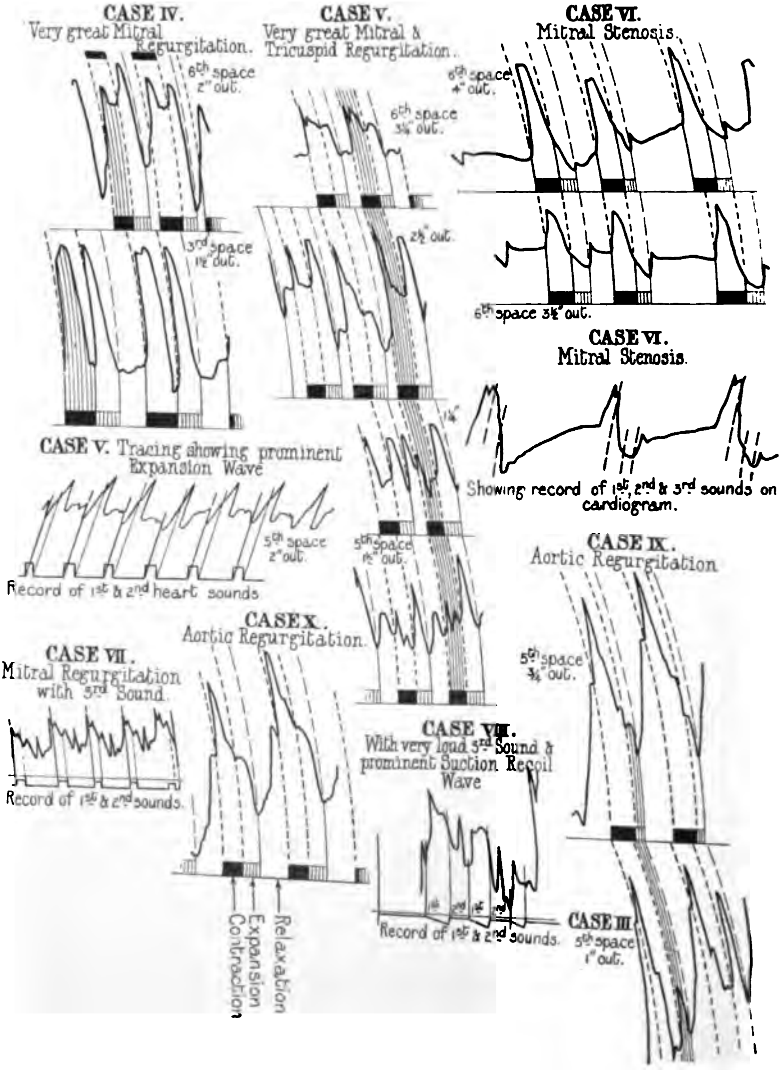
CARDIOGRAMS DEMONSTRATING THE ENLARGEMENT OF THE VENTRICLE DURING ITS EXPANSION PHASE



(For the explanation of the tracings. see opposite page.)

PLATE VIII

CARDIOGRAMS DEMONSTRATING THE ENLARGEMENT OF THE VENTRICLE DURING ITS EXPANSION PHASE


CASE IV.
Very great Mitral Regurgitation.
6th space 2" out.
3rd space 1½" out.
CASE V.
Very great Mitral & Tricuspid Regurgitation.
6th space 3¼" out.
2½" out.
1¼"
5th space 1½" out.
CASE VI.
Mitral Stenosis.
6th space 4" out.
6th space 3½" out.
CASE VI.
Mitral Stenosis.
Showing record of 1st, 2nd & 3rd sounds on cardiogram.
CASE V. Tracing showing prominent Expansion Wave
5th space 2" out.
Record of 1st & 2nd heart sounds
CASE X.
Aortic Regurgitation.
CASE VII.
Mitral Regurgitation with 3rd Sound.
Record of 1st & 2nd sounds.
Contraction
Expansion
Relaxation
CASE VIII.
With very loud 3rd Sound & prominent Suction Recoil Wave
1st
2nd
1st
2nd
Record of 1st & 2nd sounds.
CASE IX.
Aortic Regurgitation
5th space ¾" out.
CASE III
5th space 1" out.

(For the explanation of the tracings, see opposite page.)

Plate VIII

Case IV.—Very extreme Mitral Regurgitation.—The apex beat was in the sixth space in the anterior axillary line. Fig. 1 from the sixth space, ½ inch internal to the nipple line. Fig. 2 from the fifth space, 1½ inches from the sternum. They show an extremely well-marked diastolic expansion wave.

Case V.—Great Mitral and Tricuspid Regurgitation.—The apex was in the sixth space in the anterior axillary line, 4 inches from the sternum. Figs. 1, 2, and 3, cardiograms from the sixth space at distances of 3¼, 2½, and 1½ inches from the sternum. Fig. 4 from the fifth space, 1¼ inches out. Fig. 1 is of the apex type. The remainder show an extremely pronounced diastolic expansion movement. The interpretation here given was verified by very numerous observations and records of the heart sounds on the cardiogram. Fig. 5 is a tracing similar to No. 4, but taken with the polygraph and with a record of the first and second sounds on the tracing.

Case VI.—Mitral Stenosis, with a well-marked Third Sound and Mitral diastolic murmur.—The diastolic expansion movement does not show at all, but there is a well-marked wave at the commencement of the relaxation period—a suction recoil wave. Fig. 1, from sixth space, 4 inches out from sternum, and fig. 2, 3½ inches from sternum. Fig. 3, similar tracing to 1, but taken with the polygraph, and showing the position of the first, second, and third sounds, as recorded on the cardiogram.

Case VII.—Mitral Regurgitation, with a well-marked Third Sound, but no stenosis.—Tracing taken from near the apex, with a record of the first and second sounds on the cardiogram. The sounds in this case were also recorded separately, and careful measurements showed that the third sound would come just at the foot of the sharp rise which occurs between the beats.

Case VIII.—Mitral Disease, with well-marked Third Sound.—The third sound was louder than the first sound and as loud and distinct as the second sound. The time of the first and second sound is recorded on the cardiogram. There is a prominent suction recoil wave as in the preceding case.

Case IX.—Aortic Regurgitation.—Tracing from the fifth space ¾ inch from the sternum, showing absence of the diastolic expansion rise. Below this is placed for comparison a cardiogram, from a similar part of the heart, from Case III., with mitral regurgitation, where there is a prominent expansion rise.

Case X.—Aortic Reguritation.—Tracing from near the apex showing smallness of the expansion wave. Compare Case I, figs. 1 and 2; Case II figs. 2 and 3 ; and Case V, figs. 1 and 2

ESSAY XI.—ON THE THEORY OF COMPENSATION IN INCOMPETENCE AND STENOSIS OF THE MITRAL VALVE[1]

THE object of this essay is to make a clinical contribution towards the proof of what may be called the modern theory of compensation in disease of the mitral valve.

For the last fifteen or sixteen years the belief has been gaining ground that the filling of the left ventricle of the heart during diastole is mainly due to aspiration by the ventricle itself rather than to the contraction of auricle or other forces.

This view as to the filling of the ventricle, which is based upon observations such as those of Dr. H. D. Rolleston (who demonstrated the presence of a negative pressure in the ventricle during diastole), has, since 1885 or earlier, been advocated by some writers and lecturers on cardiac physiology. A very clear statement of this theory and its bearings upon the functions of the auricles will be found in some 'Lectures on the Physiology of the Vascular System,' by Mayo Collier, published 1888.

But although the physiological side of this question has for so many years been clearly stated, the new views as to ventricular aspiration do not seem to have been generally adopted by clinicians, and the necessary modifications in the theory of compensation in mitral disease have very rarely been faced in the literature of the present day.

Amongst the exceptions must be mentioned the name of Sir Clifford Allbutt, who accepts the theory in his 'System of Medicine.'

The new theory seems to be much more satisfactory than the old for many reasons. It is more satisfactory to believe that the strong left ventricle is able to compensate for its own deficiencies instead of having to fall back for assistance, as the old theory would have us believe, upon the weak right side of the heart, or the still weaker pulmonary capillaries. Also from clinical and pathological standpoints it proves more satisfactory than the old one, and I have adopted it as a working hypothesis for the last seventeen years, and have not yet found it to be at fault.

---

[1] Read before the Clinical Society of London, December 13, 1901, and printed in vol. xxxv. of their *Transactions*.

The facts brought forward in this essay are the outcome of an investigation into the loudness of the heart sounds in health and in valvular disease, but more especially as regards the loudness of the second cardiac sound in the pulmonary area.

According to the old or *vis a tergo* theory of compensation, the pulmonary second sound ought to be always more or less accentuated in mitral disease, because a very considerable portion of the work of compensation is supposed to rest upon the right ventricle. According to the new or *vis a fronte* theory, almost the entire work of compensation is done by the left ventricle itself, owing to an increase in its aspiratory power, and, therefore, according to this theory, when compensation is good the right ventricle will have no extra work to do, and the pulmonary second sound will be of normal loudness.

The result of my observations points unmistakably towards the accuracy of the latter or *vis a fronte* theory.

For the purpose of measuring the heart sounds, I found, after many experiments in obliteration of the heart sounds by the interposition between the stethoscope and the chest wall of disks of rubber, felt, sponge, &c., that the simplest and best method was to use a bag of air for cutting off the heart sounds. To do this an empty thin-walled, elastic rubber bag (a deflated toy balloon) is placed between the chest-piece of a binaural stethoscope and the chest wall, and by its inflation the stethoscope is slowly lifted up until the heart sounds are no longer heard. The height to which the particular sound is audible is measured by means of a small platform, placed between the stethoscope and the bag, which slides up and down on three legs, which are graduated in fractions of an inch, and upon which can easily be read off the height of the chest-piece of the stethoscope when the sounds are lost. The inflation can be done by the mouth through a rubber tube, which can be compressed by the lips or teeth when it is desired to maintain the fullness of the bag for a time. Very exact measurements can be quickly made by this apparatus. In the statistics which follow, I have adopted $\frac{1}{16}$ inch as the standard for measuring the distance to which the sounds are audible, and in speaking of the loudness of the sound, I shall only give the number of sixteenths of an inch at which it is still audible—e.g. a sound whose loudness I call 'sixteen' is one audible 1 inch away from the chest wall with the apparatus used. A sound of 'twenty-four' is one audible $1\frac{1}{2}$ inches away.

The observations I have made have shown that this method of measuring the sounds is most reliable, and, moreover, removes some sources of fallacy. It enables the listener to distinguish between an accentuation of the second sound due to real increase in the loudness of the valvular sound, and one due simply to the normal sound being better heard owing to the pulmonary artery being more superficial than usual. Also in several cases where there was apparently well-marked accentuation of the pulmonary second sound, I found that the sound was, in reality not audible for more than the usual distance from the chest wall, and therefore could not be said to be accentuated. In these cases it was sharper and more slapping than normal, but there was no true increase in its volume, such as might be expected if the blood tension in the pulmonary artery were raised.

I have recorded the measurements in sixteenths of an inch, and in the tables at the end of this essay the numbers given tell the number of sixteenths of an inch from the chest wall at which the particular sound referred to is audible.

In the course of the last few months, I have made over a hundred observations of all the heart sounds in some fifty patients suffering from various ailments; and in the cases of mitral disease, some twenty-six in number, I have paid special attention to the pulmonary second sound, making some 150 observations of the loudness of this sound alone.

From these observations it appears that the distance of audibility of the pulmonary second sound varies in healthy hearts from ten or twelve up to twenty sixteenths of an inch, and that slight ailments or a little excitement will increase its loudness up to twenty or twenty-four sixteenths or more.

In anæmia in young women, on the other hand, the loudness of the pulmonary second sound is apt to range much higher. This is, no doubt, partly due to the very superficial position of the pulmonary artery in these cases, but there must also be some true accentuation. Amongst some four or five cases which I have examined, I have in one case recorded a pulmonary second sound heard 48 sixteenths of an inch from the chest wall, and two others of 44 and 36 respectively.

In comparison with these very loud sounds of anæmia, heard as much as 3 inches (or $\frac{48}{16}$ inch) away from the chest wall, it is very striking to find that in disease of the mitral valve the pulmonary second sound can rarely be heard more than $\frac{22}{16}$ inch to $\frac{24}{16}$ inch away (i.e. $1\frac{3}{8}$ inches to $1\frac{1}{2}$ inches), and in only one case out of the twenty-six examined was the pulmonary second sound regularly heard as much as $\frac{26}{16}$ inch away (*vide* Table II).

The fact that there is no very marked increase in the loudness of the pulmonary second sound during the process of compensation is also shown in Table III, which shows a series of observations upon a patient with mitral stenosis with marked failure of compensation, who speedily recovered under treatment, and in whom the regular loudness of the pulmonary second sound never exceeded 22.

In one case of compensated mitral regurgitation the onset of anæmic dilatation of the right ventricle was accompanied by a rise in the loudness of the pulmonary second sound from 16 or 18, at which it usually stood, to 44.

In another case a little nervous excitement caused a pulmonary second sound, usually heard only 16 or 20 sixteenths of an inch away, to be audible at a distance of 28 inches, and once at 40 inches.

The discussion of the cause of this low average in the loudness of the pulmonary second sound would be out of place here; but the fact is interesting as showing that the pulmonary second sound is much louder is anæmia and nervous excitement than it is when the right ventricle is supposed to be working at its hardest, in order to try to overcome the embarrassment of the pulmonary circulation due to mitral stenosis or regurgitation.

I now turn to the theoretical consideration of the newer theory of compensation, in order that the bearings of my observations on the theory may be estimated ; and I have, for the sake of brevity, expressed the theory in a series of propositions, some self-evident and some debatable, and needing any support which my observations as to the loudness of the heart sounds can give.

In Mitral Incompetence

Proposition I.—The process of compensation necessitates dilatation of the cavity of the left ventricle, as well as hypertrophy of its walls, and the amount of dilatation in the compensated heart is directly proportional to the amount of blood which regurgitates into the auricle at each beat of the heart.

As mitral incompetence involves the leakage back into the auricle of a certain part of the contents of the ventricle at each beat of the heart, it therefore follows that in order to compensate for this leakage and allow of a normal amount being thrown into the aorta at each beat—

1. The ventricle must enlarge so as to hold both the normal amount and that which will escape back into the auricle.

2. It must thicken its walls so as to be able to move this additional volume of blood.

3. The theory of the enlargement of the ventricular cavity is as follows : On the first occurrence of regurgitation through the mitral valve a progressive over-distension of the auricle will theoretically result from the leakage of blood back into that chamber. As soon, however, as the size of the auricle exceeds that of the ventricle, the latter will, during its expansion, be drawing towards itself more blood than it can hold, and the momentum of the blood which cannot find entrance will tend to dilate the cavity of the ventricle. In this way the ventricle will be dilated by its own aspiratory force until its size is comparable to that of the auricle. This will not occur until the

ventricle is large enough to hold the full amount of the leakage in addition to its normal contents (*vide* Essay XII, pp. 435 and 436).

Proposition II.—The dilatation and hypertrophy, which can compensate for the defective working of the left ventricle during systole, will, according to this theory, also relieve the auricle from embarrassment during diastole by ensuring the proper filling of the ventricle.

For (1) since the enlarged ventricle is able to accommodate all the regurgitated blood in addition to its normal contents, it follows that it will be as easy for the abnormal amount of blood to enter the abnormally large ventricle as for the normal amount to enter the normal ventricle ; and

(2) the increased muscularity of the enlarged ventricle will ensure the slight addition to the aspiratory force which is required to draw the larger amount into the ventricle.

Proposition III.—Therefore, by the time the expansion of the ventricle has ceased, the auricle will have been relieved of all the regurgitated blood, and only the normal amount will remain to be dealt with by it.

Proposition IV.—Therefore, in *fully compensated* mitral regurgitation, no extra work will be thrown upon the left auricle, and there will be no embarrassment of the pulmonary circulation and no accentuation of the pulmonary second sound.

In Table IV are given the details of five cases of compensated mitral regurgitation : two of them, Cases IV and V, rather severe cases, and the others well-marked cases. In each of these it will be seen that the average loudness does not exceed 17, which is normal.

Proposition V.—Neither would there be any dilatation of the left auricle unless the regurgitation were extreme.

Since the systole of the auricle and of the ventricle together occupy one-half of the cardiac cycle, it follows that only one-half of the full volume of blood will normally enter the auricle during this portion of the cycle. Therefore, there will be room in the auricle during the ventricular systole for any amount of regurgitated blood up to one-half of the total quantity put into circulation at each beat. If, on the other hand, the amount of regurgitation were to exceed one-half of the normal output of the ventricle there might be dilatation of the auricle, even when compensation was fully established.

Proposition VI.—According to this theory of compensation there ought to be a slight increase in the loudness of the aortic part of the second sound, because the greater expansile force of the hypertrophied and dilated ventricle will induce a more powerful recoil of the blood in the aorta, and will result in a more powerful closing and stretching of the aortic valves than in the case of the normally acting ventricle.

IN MITRAL STENOSIS

According to this theory, the left ventricle increases its aspiratory power in order to facilitate the entry of blood through the narrowed orifice, and so establish compensation. This is brought about as follows:—

Proposition VII.—In mitral stenosis the left ventricle contracts with greater force than normal in order that the rebound, both muscular and elastic, may be correspondingly increased.

1. An evidence of this is the increased loudness of the first sound which occurs in mitral stenosis.

This is shown in Table III, which deals with Case III. Before the establishment of compensation the first sound is seen to be 32, sometimes reaching 40; and after compensation is established it is 22 or over, which is above the normal.

Very loud first sounds are also seen in Cases VII and VIII of Table V and Case X of Table VI, which are severe cases of mitral stenosis. Also in Table VI the first sound in the five cases with good compensation averages 20, as compared with an average of about 12 in the five cases of mitral regurgitation.

2. Temporary embarrassment of the circulation is accompanied by an increase, often excessive, in the loudness of the first sound, which lessens again as the circulation improves (*vide* Tables III and V).

This is well shown in Case VII, Table V, where the loudness of the first sound is suddenly increased from 36—a sound inaudible with the stethoscope $\frac{1}{16}$ inch from the chest wall if no conductor be used—to a sound audible without the stethoscope at a distance of 2 feet from the chest. The same increase to a less extent is seen in Cases VIII and IX, and is often noticeable.

3. The increase in the force of the ventricular contraction is also suggested by the occurrence of the hypertrophy of the left ventricle which is not infrequently noticed in mitral stenosis.

Proposition VIII.—The left ventricle alters its beat. Its contraction terminates with greater suddenness and force than normal. This concentration of ventricular effort at the termination of the systole has the effect of further increasing the force of the ventricular recoil which immediately follows the systole, lasting as it does only $\frac{1}{10}$ of a second.

1. This is the explanation of the sharp and loud end of the first sound which is so characteristic of mitral stenosis.

2. The characteristic cardiograms of mitral stenosis often show a more sudden recession of the heart from the chest wall at the end of the systole than in the normally beating heart.

Proposition IX.—As a result of this additional aspiration, the auricle can be relieved of much, if not all, of the additional work which the narrowness of the mitral valve entails. We find very frequently an absence of dilatation of the left auricle in mitral stenosis in cases where compensation was perfect at the time of death (*vide* Dr. Samways, *British Medical Journal*, vol. ii, 1898, p. 365).

Proposition X.—Therefore, in compensated mitral stenosis, if the narrowing be not very excessive, no extra work is thrown upon the right side of the heart, and there is no engorgement of the lungs.

1. As evidence of this, we find the pulmonary second sound of normal loudness, even in cases of severe mitral stenosis (*vide* Table VI and Table V, Cases VII and VIII, in part).

2. We find that the accentuation of the pulmonary second sound, which is characteristic of failing compensation, gradually lessens as compensation improves (*vide* Table II).

3. We may find no clinical evidences of pulmonary congestion or engorgement, even in severe cases of mitral stenosis.

Proposition XI.—We may also expect some increase in the loudness of the aortic second sound, apart from that due to increased arterial pressure, because of the increased ventricular aspiration, as suggested in Proposition VI.

In conclusion, although I am not able to bring a very large number of observations, owing to the amount of time required to make the careful observations here recorded, I nevertheless hope that I have brought forward enough evidence to show the desirability of making wider investigations with regard to this important question.

The importance of this question, so far as treatment is concerned, is obvious; for if the expansion movement does play an important part in compensation, we are greatly handicapped as regards the treatment of our patients by our almost entire ignorance as to the effect of drugs upon that important expansion movement. A recognition of our need of having cardiac tonics classified into those which increase the expansile force of the ventricle and those which do not will be the first step towards having that need supplied.

SUMMARY OF THE TABLES, SHOWING THE LOUDNESS OF THE HEART SOUNDS IN HEALTH AND IN VALVULAR DISEASE

The figures given show the distance, in sixteenths of an inch, to which the sound is audible with the apparatus used (e.g.

a pulmonary second sound of '16' is one audible at 1 inch distance; one of '40' is one audible at 2½ inches from the chest wall). The figures given in inches and fractions (e.g. '= ½″') show the greatest distance at which the sound is audible by a binaural stethoscope when no conductor is used.

Details of cases summarised in this table	Pulmonary area		Aortic area		—
	2nd sound	1st sound	2nd sound	1st sound	
I. Averages of—					
Six normal hearts . .	16	14⅓	14½	11	Table I.
Four normal hearts .	10	16	11	14	
Five hearts in slight anæmia, rheumatism, &c.	20	26	20	21	
Three cases of extreme anæmia, &c.	40	29	19	15	—
II. Cases with compensated mitral regurgitation (average loudness [*see* Table IV.]):					
Case II . . .	17	12	13	7½	9 observations.
Case III . . .	16	10	11	12½	6 ,,
Case IV . . .	7	7	5	6	2 ,,
Case V . . .	16	18	22	15	5 ,,
Case VI . . .	16	10	9	6	3 ,,
III. Cases with compensated mitral stenosis (*see* Table VI):					
Case X . .	10	21	8	10	4 ,,
Case XI . .	14	19	8	13	2 ,,
Case XII . .	19	20	23	16	3 ,,
Case XIII . .	13	20	14	20	2 ,,
Case XIV . .	16	20	17	23	4 ,,
IV. Cases with failing compensation in mitral stenosis:					
Case I . .	8	48=½″	14	36	1 observation.
Case VIII . .	23	70=¾″	8	30	1 ,,
Case VII . .	24	2 feet	26	48	
Case X . .	22	44	—	—	—

TABLE I

Table showing measurements of the loudness of the normal heart sounds in the pulmonary and aortic areas respectively.

The figures given in this and the following tables are measurements in sixteenths of an inch of the distance from the chest wall at which the sound is audible with the apparatus used. Figures in parenthesis (16) are distances at which the sound in question is *sometimes* heard. Figures in square brackets [16] show the distance at which, in an irregularly beating heart, the sound may very occasionally be heard.

Sex	Age	Pulmonary area		Aortic area	
		Second sound	First sound	Second sound	First sound
F.	49	6	16, 18	10, 12	16
F.	49	12, 9, 12	22 (24)	16	20 [24]
M.	51	10, 10, 12	14, 16	10	12 (16)
M.	36	10, 10	16, 16	8	9 (12)
M.	36	9, 9	16, 16, 15	8	12
F.	19	16	10, 11 (12)	16	12
F.	23	12 (14)	16	14 (16)	14 (16)
F.	21	17 (22)	15, 20 [22]	14 (18)	12 [18]
M.	34	14	11	7	4
M.	41	16, 14	12	15	12
F.	44	16	20	15	12
F.	23	16 (18)	12 (18)	20	12 (14)
Average .		13½	16·2	14·3	12

TABLE II

Table showing a classification of 150 observations upon the loudness of the pulmonary second sound in twenty-six cases of mitral incompetence and stenosis, according to their loudness, and also to their occurrence in patients with fully established compensation, or in patients treated for failing compensation. This second group includes observations made on patients after the establishment of compensation. In the uncompensated group in some nine instances feeble sounds were due to feebleness of the ventricle and not to the establishment of compensation. For the sake of completeness, I give in a second column, enclosed in brackets, the number of 'occasional' observations made in addition to the regular ones.

Degree of loudness measured in sixteenths of an inch	Number of observations made in fully compensated hearts	Number of observations made in uncompensated hearts
0 to 9	4 and (1)	2 and (1)
10 to 14	3 and (3)	18 and (2)
15 to 17	11 and (1)	9 and (2)
18 to 19	3 and (0)	10 and (0)
20 to 24	4 and (3)	37 and (19)
25 and over	0 and (1)	5 and (12)
	25 and (9)	81 and (36)

TABLE III

CASE I.—Measurements of the loudness of the heart sounds in a case of mitral stenosis with extreme dilatation of the right ventricle and dropsy, showing the variations taking place during the restoration of compensation.

On the date of the first measurement the relative cardiac dullness extended 2 inches to the right of the sternum in the fourth interspace, and the apex was in the sixth left interspace some 5 inches from the sternum—i.e. in the

Date	Pulmonary area		Aortic area		Condition of the patient
	Second sound	First sound	Second sound	First sound	
23, viii	8	32 (40) [48]	14	36 (24) [44]	Very ill.
25, viii	22, 22	24 (28)	20, 21	22 (28)	Much better.
26, viii	20 (22) [32]	22 [32]	16 (20)	28	Improving.
28, viii	16 [17]	22	10 (12)	22	,,
4, ix	18 [22]	22	16, 16	22	,,
4, ix	20	—	18	—	,,
20, ix	20 [22]	24	20	22	Got up on the 18th.
29, ix	20, 19 (22)	22 (24)	25	25	
29, ix	22 (24)	23	22	23	
29, ix	24	24	24	25	

NOTE.—On 23, viii, the pulmonary second sound seemed loud and slapping, but could not be heard more than half an inch away from the chest wall. On 29, ix, the loudness of the sounds last noted was probably in part due to nervousness produced by a prolonged examination.

anterior axillary line. At the time of the last measurement the apex was in the fifth left interspace, 4 inches from the sternum, and the patient was walking about with very little dyspnœa.

TABLE IV

Measurements of the cardiac sounds in compensated mitral regurgitation.

Case	Date	Pulmonary area		Aortic area	
		Second sound	First sound	Second sound	First sound
Case II, F., æt. 15	3, viii	12, 16, 24	8, 14, 14	9	8
	5, viii	14, 20	8, 10	14	2
	10, viii	16, 16, 18	10, 10	18	6
	4, ix	18 (20)	28	10	14
Case III, M. .	6, viii	16, 14	8, 10	14	8, 12, 14
	7, viii	12	10	10	8
	10, viii	18, 18	10, 12, 12	10, 12	14, 16
	14, viii	18 [22]	9	10	15
Case IV, M., æ. 26	8, viii	4, 6	4, 6	6	6
	29, viii	10	13	4	7
Case V, F., æt. 33	22, viii	16, 14[1]	12	22	16
	5, ix	20 [28]	18	22	14
	19, ix	15, 15	24	21	14
Case VI, F. .	23, viii	17, 18	8	10	4
	26, viii	14 (20)	12	8	8

---

[1] At this date the pulmonary second sound seemed much louder and sharper than the aortic, but on testing it with the apparatus used it was found that the aortic second sound had more penetrative power than the pulmonary.

TABLE V

Cases of mitral stenosis illustrative of the lessening of the loudness of the first sound as compensation improves, and vice versa.

Case	Date	Pulmonary area		Aortic area		Condition of the patient.
		Second sound	First sound	Second sound	First sound	
Case VII, æt. 31 . .	8, viii	16	{1$\frac{1}{2}$ feet away}	14	48 +	Very breathless.
	10, viii	16	24 (48)	20	—	Improving.
	23, viii	17, 18	48 + {$\frac{1}{8}$–$\frac{1}{4}$ in.}	10, 12	32	„
	27, viii	20 (22)	40 (48), {$\frac{1}{16}$ in.}	22 (24) (32)	40	„
	30, viii	14	36	14 (16)	40	Got up on 30, viii.
	4, ix	24 (28) [32]	{2 feet away}	24, 28	48 +	'Much upset.'
Case VIII, very severe case	23, viii	22	70 ± {$\frac{3}{4}$ in.}	8	24 to 28	
	25, viii	24 (20) [32]	50 + {$\frac{1}{4}$–$\frac{1}{2}$ in.}	8 (12)	30	
	27, viii	22 (24)	32 {$\frac{1}{16}$–$\frac{1}{8}$ in.}	14	36	
	4, ix	14 (16)	28 (30)	8	28	Better.
Case IX, æt. 33, very severe case .	29, viii	14	26	10 [12]	20 (22)	Breathless.
	31, x	20	18, 20	8	12, 16	Much better.
Out-patient, compensation fair	5, xi	20 (22)	24	16	23	Pulse 100; rather breathless after walking.
	5, xi	24	22 (24)	22 (23)	16 (22)	Pulse 80; better again.

Figures in braces { } refer to observations made without the use of the rubber bag.
See also Table VI, Case X, last few observations.

TABLE VI

Table showing the loudness of the heart sounds in compensated mitral stenosis.

Case	Date	Pulmonary area		Aortic area	
		Second sound	First sound	Second sound	First sound
Case XI, mitral stenosis and double aortic murmur	11, viii	16 (20)	20 [26]	8	14 [18]
	26, viii	12 (14)	18 [20]	8	12 [18]
Case XII, F., æt. 42	15, viii	20, 22	16, 22	24, 25	20
	22, viii	14 [16]	16, 22	20	13
Case XIII, M., æt. 33, a severe case	24, ix	8 (12)	17	16	17, very well.
	7, xi	19	22	12	24, breath bad.
Case XIV, M., æt. 40	19, ix	16, 16 (22)	23	18 (22)	26
	31, x	16, 16	18	16 (18)	21
Case X, F., mitral and aortic	3, viii	8, 12	20, 22	8	10
	5, viii	10, 12	22	0 (murmur)	10
Case X, Compensation failing again	10, viii	24 (32)	32, 28 (24)	0 (murmur)	Murmur.
	14, viii	22 (28)	44	—	—
	4, ix	12 (14)	48 ($\frac{1}{16}$ in.)	—	—

ESSAY XII.—ON THE DIASTOLIC CARDIAC SOUND WHICH CAUSES SPURIOUS REDUPLICATION OF THE SECOND SOUND AT THE APEX, AND IS SOMETIMES CALLED THE THIRD SOUND OF THE HEART[1]

THE object of this essay is to record several cases in which this sound was well heard, because I think they throw important light upon the mode of occurrence and the clinical value of this diastolic cardiac sound.

But before describing the cases, I must discuss briefly what I believe to be the cause and the meaning of this sound. I take what I consider to be the generally accepted view, that this spurious reduplication of the second sound is due to the appearance of an additional sound after the true second sound, and that this sound is in some way produced by the entry of blood into the ventricle at the commencement of diastole. This view was ably advocated twenty years ago by Dr. Sansom, and is well supported by arguments in his book on the 'Diagnosis of Diseases of the Heart.' I need not speak of the clinical features of this sound: they have been often described—but nowhere, in my opinion, more clearly and accurately than by Dr. Arthur Phear in the *Lancet* for 1897, p. 97. With him, I agree that so definite and distinct a sound ought to have a name of its own. It has been called 'the third sound of the heart,' and I do not think a more appropriate name could be found for it, and for the sake of clearness and brevity I must ask to be allowed to call it 'the third sound of the heart' in this essay.

The sound to which I refer in this essay is a short, sharp sound. I do not refer to the mitral diastolic murmur.

---

[1] Read before the Clinical Society of London, November 9, 1900, and printed in vol. xxxiv. of their *Transactions*.

The theoretical part of this paper involves so many points upon which proof may rightly be asked for, that I have found considerable difficulty in condensing all I have to say into the space at my disposal. The ground is, in part, covered by a paper upon the diastolic expansion movement of the ventricles which I read at the Ipswich meeting of the British Medical Association (*vide* Essay X). For the sake of brevity, I have put the theoretical part of my paper into the following series of propositions :—

1. The diastolic expansion of the ventricles plays so important a part in the circulation that it must be reckoned as a distinct phase of the cardiac cycle (*loc. cit.*).

2. Record of the second cardiac sound on the cardiogram shows that this sound occurs at or just after the commencement of this expansion movement (*vide* Cardiograms, Case II, figs. 1 and 2; Case V, figs. 12 and 13; and Case VI; also Plate XII at end of book).

3. Record of the so-called third sound on the cardiogram, and careful measurement of the heart sounds and tracings show that the third sound occurs at or just before the commencement of the period of muscular relaxation (*vide* Cardiograms, Cases III, V, and VI).

4. Therefore the third sound of the heart occurs at the end of the expansion movement, and marks the commencement of the third phase of the ventricular cycle. It therefore resembles the first and second sounds in marking the commencement of a distinct phase.

The first sound occurs at the commencement of the ventricular contraction.

The second sound occurs at the commencement of the ventricular expansion.

The third sound occurs at the commencement of the ventricular relaxation.

5. The duration of the expansion movement is usually about one-tenth of a second—i.e. from about half to two-thirds as long as the ventricular systole (see Table of Measurements on p. 429).

6. In cases where the third sound is well marked there is often to be seen in the cardiogram a short sharp rise, imme-

diately following the fall due to the muscular relaxation. This oscillation I propose to call (from my belief as to its origin) the suction recoil wave.

7. This wave varies somewhat in character :—

(*a*) It may be only or mainly visible in tracings taken at the apex, and may be more or less due to a tilt forward of the relaxed anterior wall of the heart, such as might be caused by the impact against it of a current of blood entering the ventricle from the auricle.

(*b*) In other cases it is disproportionately greater the more rapid the previous fall of the lever, and might be largely mechanical or of the nature of a rebound of the lever. (As this rebound does not occur to the same extent in the normal heart, or in simple mitral regurgitation, it seems to have some

MEASUREMENTS OF PHASES OF THE CARDIAC CYCLE

Measured in twentieths of a second.

Case	Measurements of sounds[1]						Measurements of cardiogram				
						Average					Average
CASE II . .											
Systole . . .	3	3½	3	2½	—	—	4	4½	4	4½	4·2
Expansion . .	—	—	—	—	2	—	2	2	2	1¾	1·96
Total beat . .	10	11½	10½	9½	9	—	11	11	10	10	10·48
Number of beats measured	—	—	—	—	—		7	7	10	5	
CASE III											
Systole . . .	3½	3½	—	—	—	3½	3¾	4	4½		4·15
Expansion . .	—	—	2½	2	1⅘	2	2	2	2		2
Total beat . .	12	12	12	11	11	11·6	12	11	11½		11·4
Number of beats measured	7	13	6	7	7		6	11	12		
CASE V											
Systole . . .	5	5	—	—		5	5½	6			5·8
Expansion . .	—	—	2	1½		1·8	1½	2			1·8
Total beat . .	14	12	14	12		13·2	12	14			13·2
Number of beats measured	8	5	5	4				6	9		

---

[1] The time between the first and second sounds is reckoned as the systole, although the first sound occurs just after its actual commencement. The time between the second and third sounds gives the duration of the expansion movement.

pathological significance, and to imply an abnormal excess of intraventricular tension at the moment when it occurs.)

(*c*) In other cases this suction recoil wave is observable in tracings taken from every available part of the ventricle wall, and is evidently due to a sudden increase in the volume of the ventricle, such as could only occur by the sudden entry of blood with considerable force (*vide* Cases II, III, and IV).

8. In some cases, therefore, where this third sound occurs, we seem to have evidence of some increase of tension within the ventricle just after the onset of ventricular relaxation, and sometimes we see an actual general expansion of the ventricle at this point of time.

9. The cardiograms show that this increase of tension is due to the preceding activity of the ventricles, for the suction recoil wave is separated from the following auricular systole by an interval which varies with the varying length of the long pause (*vide* Cases I and III).

10. We may therefore conclude that this distension of the ventricle is due to the entry into it of blood which was set in motion by the ventricular aspiration developed during the preceding expansion movement. (The other alternatives would be to believe in a second ventricular systole in the middle of the long pause, or to believe in an early and wholly unprecedented form of auricular activity. There is no evidence at all in favour of either of these.)

11. We therefore can say that where the third sound is well marked there is evidence that blood is still flowing into the ventricle in a more or less powerful stream after the relaxation of its muscles has set in.

12. It will, I think, be readily admitted that in the case of the normal heart the size of the mitral orifice is so considerable, in comparison with the cubic capacity of the auricle, that a powerful ventricular aspiration, lasting half as long as the systole, would suffice to draw all the contents of the auricle into the ventricle by the time the aspiration ceased.

Therefore, we may say that in the normal heart all movement of blood due to ventricular aspiration will practically have ceased by the time relaxation occurs.

13. As a result of the above considerations, I have ad-

vanced the following suggestions as to the mode of production of this sound.

I. The third sound of the heart is not produced by the first inrush of blood into the ventricle. (In some cases of mitral stenosis, I believe that a sound or a murmur may be produced at the mitral orifice by this first inrush; but it is, of course, synchronous with the second sound.)

II. During the expansion movement the segments of the mitral valve are, presumably, kept tense by the activity of the expanding muscles, but on the onset of relaxation they are free to move. If, therefore, a sufficiently powerful stream of blood is still flowing through the mitral orifice at the onset of relaxation, it will be apt to move the valve segments with an audible sound at this point of time.

III. If my view is correct, the occurrence of a third sound is evidence that the auricle is unable to empty its contents into the ventricle as rapidly and completely as normal.

IV. This prolongation of the flow will occur—

(1) Where there is a narrowing of the mitral orifice; or—

(2) Where the auricle holds more blood than the ventricle can draw through the normal mitral orifice during the expansion movement.

V. We may, therefore, have a third sound, occurring—

(1) In mitral stenosis.

(2) Where the auricle is so dilated as to hold more blood than the ventricle does.

(3) Also, probably, where the muscle of the ventricle is too weak to develop an amount of aspiration sufficient to empty the auricle as rapidly as normal.

The cases to which I wish to refer belong mostly to the second of the three last-named groups, but I shall first show two cardiograms from the apex of a case of ordinary mitral stenosis with a well-marked third sound. They both show (as is usual in mitral stenosis) an absence of any expansion at the time of occurrence of the second sound (compare Case V, fig. 153). After the fall of the lever which marks the onset of relaxation there is a sudden sharp rise, followed by a second or a third. These are well marked in fig. 143, which was taken on the outer side of the apex, but when the heart was acting excitedly.

On this tracing I did not record the heart sounds. I have not marked the position of the third sound on this cardiogram, but it would occur at the foot of the fall just before the recoil waves.

EXPLANATION OF THE PLATES

The tracings here shown were all taken with a Galabin's cardiograph, and are to be read from left to right.

In all the tracings except Case IV, figs. 148 to 151, and Case V, fig. 154, the position of the cardiac sounds as marked on the cardiogram was ascertained by synchronising the raps of an electric signal with the heart sounds, and in this way obtaining a simultaneous record of the heart sounds and the cardiogram. The actual record of the heart sounds is shown in relation to the cardiogram in Case II, fig. 144; Case IV, fig. 152; and in Case V, fig. 153. In all other cases the complete record is not shown for want of space.

Enlarged photographs of the tracings in question were, however, shown to the members of the Society when this essay was read before them, and the accuracy of the interpretations here given was demonstrated.

CASE I.—MITRAL STENOSIS WITH A WELL-MARKED THIRD SOUND

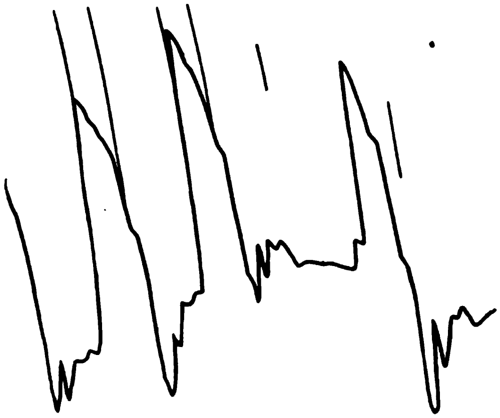


FIG. 142.—TRACING TAKEN AT THE APEX, WHICH WAS IN THE FIFTH INTERSPACE, 2½ INCHES FROM THE STERNUM.

Record of the first and second sounds on the cardiogram. In the case of the third beat the sound record is faulty owing to the irregularity of the beat. The third sound would occur at or just before the lowest point of the fall after the second sound. The suction recoil waves following the fall are well shown, and their relationship to the long pause demonstrates them to be of ventricular and not auricular origin.

In Case IV, figs. 148 to 151, the theoretical position of the first, second, and third sound has been marked.

In Case V, fig. 154, the theoretic position of the second sound as marked in the figure was verified by other combined tracings from the same case.

CASE I.—MITRAL STENOSIS WITH A WELL-MARKED THIRD SOUND

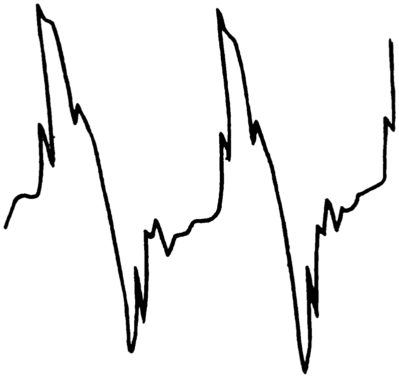


FIG. 143.—TRACING SIMILAR TO FIG. 142, BUT TAKEN FROM NEAR THE APEX AND WHEN THE HEART WAS ACTING EXCITEDLY.

The suction recoil waves are higher than in fig. 142. The sharp wave just before the main systolic rise is probably of auricular origin.

CASE II.—This is the case to which I wish to draw special attention (*vide* pp. 397, 407).

It was the case of a girl aged twelve, admitted to the Birmingham General Hospital suffering from pericarditis and rheumatic pains, with a history of acute rheumatism eleven weeks previously.

On admission, she was a pale, poorly nourished child, with considerable dyspnœa, but no dropsy. There was well-marked pericardial friction all over the cardiac area, and a loud mitral systolic murmur, which was well conducted to the axilla and back. It was specially loud posteriorly in the left mid-interscapular area, and was well conducted down the spinal column, being audible as far as the sacrum. The apex was, however, not displaced outwards or downwards, being in the fifth interspace in the nipple line. There was a loud third sound. The aortic valves were healthy. There was a slight amount of tricuspid regurgitation with some pulsation of the veins of the neck, also some slight enlargement of the liver, but no pulsation. There was no marked dilatation of the heart to the right, and none of the ordinary signs of failure of the right side of the heart.

She remained in much the same state for a month or more, confined to bed with considerable dyspnœa and marked anæmia. The increase of the anæmia was accompanied by marked upward dilatation of the right ventricle, and the shock of the pulmonary valve closure could be felt high up in the second interspace some 1 inch from the sternum.

Case II.—Mitral Regurgitation with a Well-marked Third Sound (as well as the Systolic Murmur)

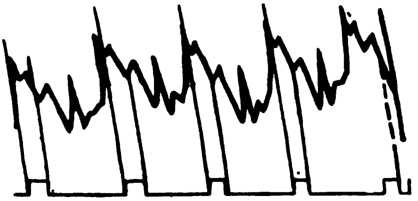


Fig. 144.—Record of the First and Second Sounds on a Cardiogram from near the Apex. (Taken in the Fifth Interspace, 2 inches from the Sternum.)

Note the prominent and pointed suction recoil wave occurring some time after the second sound.

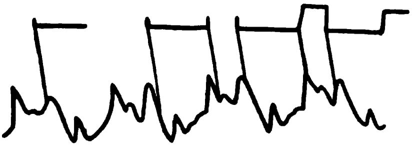


Fig. 145.—Tracing taken from the Third Left Interspace, 1½ inches from the Sternum.

The timing of the sounds is not quite accurate. The positions of the first and second are recorded. Note the presence of a suction recoil wave in this tracing taken over the conus arteriosus of the right ventricle.

After about five or six weeks she began to show signs of ulcerative endocarditis, and she also began to have attacks of anginal pain.

Throughout the case the symptoms were more those of aortic disease with failing forward pressure than of the ordinary mitral type, and it was clear to me that something was preventing the ordinary process of compensation from taking place.

The anginal attacks were very similar to the ordinary ones of aortic disease, but of no very great severity, and not accompanied by any great excess of dyspnœa, nor by the usual sense of dread. She gradually got worse, and died rather suddenly of simple cardiac failure. There was at no time any dropsy or engorgement of veins, nor much cyanosis.

Cardiograms taken from any part of the heart showed an extremely well-marked suction recoil wave. In those taken at or near the apex there was a sharp-pointed wave, just following the cessation of ventricular activity, which rose as high as the systolic eminence itself (*vide* figs. 144 and 145). The third sound would come in the hollow just before this recoil wave.

The diagnosis was that there was very considerable mitral regurgitation, with a dilated left auricle, because of the loudness of the heart sounds in the interscapular area and down the spine ; also in view of the loudness of the third sound (and the presence of an occasional diastolic murmur) mitral stenosis was suspected. The pericardium was probably adherent.

At the post-mortem there was, however, no narrowing of the mitral orifice. It was of normal size, but its cusps and chordæ were thickened, and it was evidently incompetent. There were recent small vegetations on the aortic and mitral valves. In spite of the regurgitation the ventricle was of normal size, its cavity holding 90 c.c. ; the left auricle was, on the other hand, very greatly dilated, holding 164 c.c. The other chambers were not specially dilated.

The explanation of the case is, I believe, as follows : The pericarditis preceded the mitral disease, and the support given by the adhesion of the pericardium prevented the left ventricle from dilating sufficiently for compensation to become established. It also prevented the right side from failing, and therefore great strain was thrown upon the left auricle, causing its dilatation. The pallor, dyspnœa, and absence of dropsy were due to the small amount of blood which the heart could put in circulation, losing, as it did, a considerable portion of a normal charge at each beat, owing to the regurgitation.

This case gives us a clear demonstration of the process of compensation when we read the clinical signs and the cardiograms in the light of the post-mortem.

We see the reason for the extreme suction recoil wave. We have a ventricle holding only 90 c.c., aspirating with all its force at an auricle holding 164 c.c. At the end of the expansion movement of the ventricle, when the ventricle has, in virtue of its elastic and its muscular recoil, drawn all the blood into itself which it can hold, there is still nearly as much again in the auricle waiting to come in. This has already been set in motion by the ventricular aspiration, and its momentum carries it on towards the ventricle, causing its dilatation, and making the sudden rise in the cardiogram. If the walls of the ventricle had not been unduly resistant (owing to the pericardial adhesion) this distensile force would long ago have dilated the ventricle till it was large enough completely to empty the auricle during its expansion movement, and so re-establish normal conditions. It could not do so, however. The strong rush of blood into the ventricle after the onset of relaxation would be amply sufficient to cause the loud third sound, heard during life, and the prominent recoil wave in the cardiogram.

This case has given me a clear demonstration of the means whereby compensatory dilatation of the ventricle is established in mitral regurgitation, and I feel much gratitude to that little pointed recoil wave in the cardiogram.

Case III.—The next set of tracings are from a child who had pericarditis, and whose symptoms were very similar to Case II, but whose heart was greatly dilated. There was a systolic mitral murmur, a well-marked third sound, and mid-diastolic murmur.

The suction recoil wave was most prominent here. She probably had a dilated left auricle, as in Case II. She died of sudden heart failure, but unfortunately no post-mortem examination could be obtained.

In fig. 146 the first and second sounds are recorded on the cardiogram, and in fig. 147 the second and third sounds only.

CASE III.—MITRAL REGURGITATION WITH A WELL-MARKED THIRD SOUND

The apex was in the seventh interspace in the axilla, about 4½ inches from the sternum.

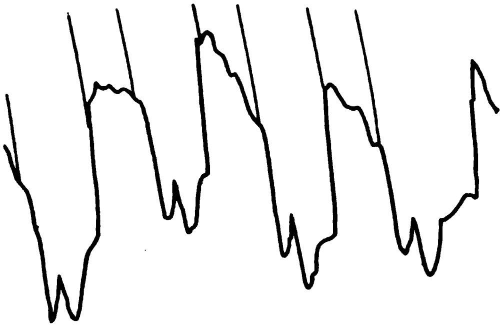


FIG. 146.—FROM THE SIXTH INTERSPACE, 4½ INCHES FROM THE STERNUM

A record of first and second sounds on the cardiogram is shown. The suction recoil wave is seen after the diastolic fall of the lever of the cardiograph.

A very prominent suction recoil wave is seen in tracings taken from all parts of the left and right ventricles.

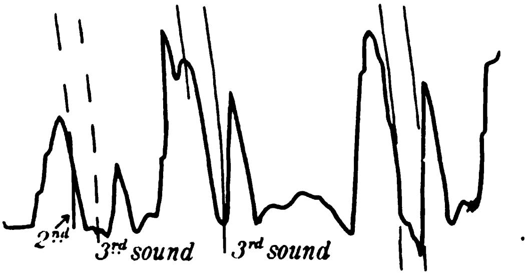


FIG. 147.—FROM THE SEVENTH INTERSPACE 4 INCHES FROM THE STERNUM (I.E. NEARLY 1 INCH INTERNAL TO THE APEX BEAT).

Record of second and third sounds on the cardiogram is given. Note the very high suction recoil wave which follows the third sound. The timing of the third beat is not quite accurate owing to the irregularity of its interval after the preceding beat.

CASE IV.—The next set of tracings are from a woman, aged forty-six, who suffered from palpitation, swelling of the legs, and occasional dyspnœa. The interesting feature about the case was that the third sound was much louder than the first sound. The heart was extremely difficult to time, because the attention was at once arrested by the second and third sounds, and they appeared to be first and second. There was a faint presystolic murmur and a faint first sound.

CASE IV.—PATIENT WITH A VERY LOUD THIRD SOUND, A FAINT FIRST SOUND, AND A VERY FAINT PRESYSTOLIC MURMUR

A very prominent suction recoil wave is seen in tracings taken from all parts of the left and right ventricles.

FIG. 148.

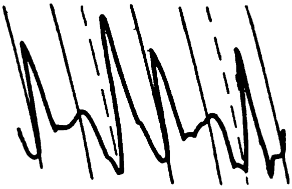


FIG. 149.

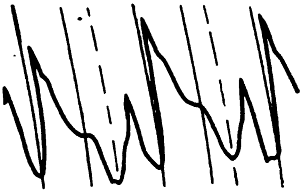


FIG. 150.

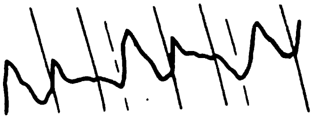


FIG. 151.

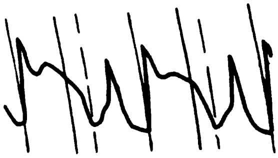


FIG. 148.—TRACING TAKEN AT THE APEX (IN THE FIFTH INTERSPACE, 4 INCHES FROM STERNUM).

FIG. 149.—TRACING TAKEN IN THE FOURTH INTERSPACE, 1 INCH FROM STERNUM.

FIG. 150.—TRACING TAKEN IN THE THIRD INTERSPACE, 2½ INCHES FROM STERNUM.

FIG. 151.—TRACING TAKEN IN THE EPIGASTRIUM.

In these tracings the approximate theoretical position of the heart sounds are shown. The position of the third sound is marked by a broken line.

On taking cardiograms, it was found that there was an extremely prominent recoil wave. In some parts of the heart it was the most prominent part of the cardiogram. It was present in all tracings. I show tracings from the apex, the fourth interspace 1½ inches out, the fifth space 3½ inches out, and from the epigastrium, and also one upon which I succeeded in recording the time of occurrence of the first and second sounds upon the cardiogram. The timing is sufficiently accurate to show that the third sound would occur in the hollow just before the suction recoil wave, as in fig. 147.[1] The explanation of this recoil wave appearing in tracings taken over the right ventricle is probably to be found in the movement of the interventricular septum.

CASE IV

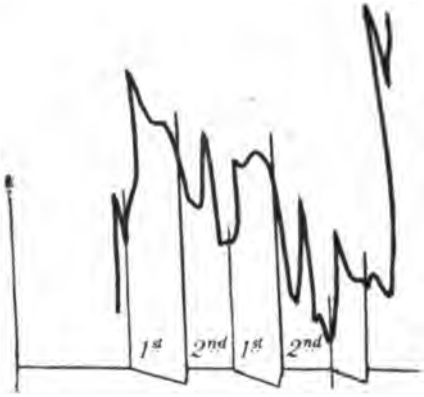


FIG. 152.—RECORD OF THE FIRST AND SECOND SOUNDS ON THE CARDIOGRAM.

Note the prominent suction recoil wave occurring some time after the second sound. The irregularities of the tracing are due to respiratory movements of the chest.

CASE V.—I show two tracings from a woman with compensated mitral regurgitation with some stenosis. The tracings show the prominent expansion movement characteristic of mitral regurgitation. There was also in this case a well-marked third sound, and a mid-diastolic murmur following it.

In this case the third sound occurs just before the foot of the fall of the apex tracing, and its place is often marked by a small oscillation in the downstroke. I would suggest, as an explanation of this, the possibility that the musculi papillares relax slightly before the rest of the ventricle wall, and so admit of the third sound occurring just before the onset of general relaxation.

Fig. 153 is a cardiogram taken just internal to the apex with a record of the second sound marked on it. It shows the systolic depression in the cardiogram, followed by a well-marked

---

[1] The size of the suction recoil wave in these tracings would suggest the possibility of its being due to an extra systole; but there was nothing in the character of the sounds to suggest this and, moreover, the rise shown in Case III is nearly as great as in this one, and there could be no doubt as to the sound being a true third sound in that instance.

CASE V.—MITRAL REGURGITATION AND STENOSIS WITH A WELL-MARKED THIRD SOUND

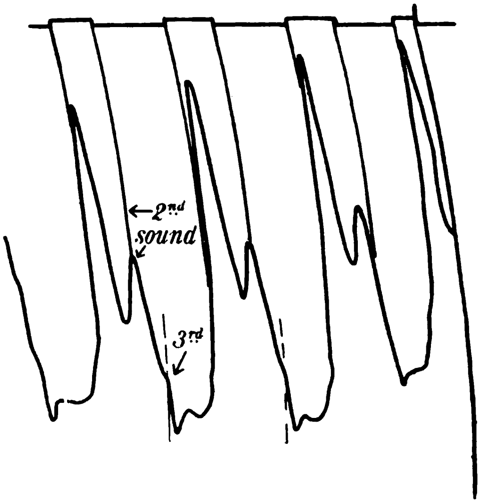


FIG. 153.—TRACING FROM THE APEX, SHOWING A RECORD OF THE FIRST AND SECOND SOUNDS ON THE CARDIOGRAM, AND THE THEORETICAL POSITION OF THE THIRD SOUND AS VERIFIED BY OTHER TRACINGS.

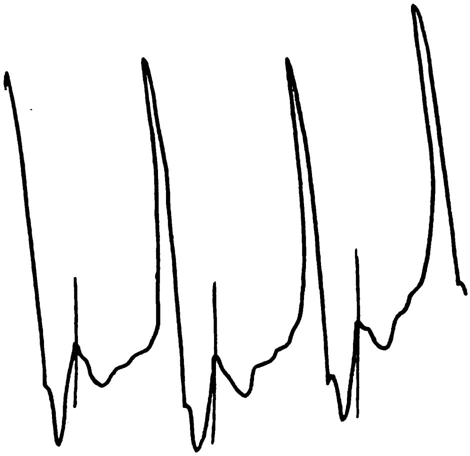


FIG. 154.—TRACING TAKEN INTERNAL TO THE APEX WHERE THE DIMINUTION IN THE VOLUME OF THE VENTRICLE AT THE END OF THE SYSTOLE CAUSES A FALL IN THE TRACING.

The position of the second sound is shown and the rise due to the diastolic expansion movement is seen. The third sound would occur in the following notch.

expansion wave. The second sound occurs near the top of this wave.

Case VII shows a cardiogram from a case of anæmia with a well-marked third sound, and is a record of the second and third sounds on the cardiogram. It shows the third sound coinciding with a small oscillation near the foot of the diastolic fall of the tracing.

Before concluding this essay, I must express my indebtedness to my house-physician, Dr. Cyril Lewis, whose patience and skill in manipulating the cardiograph while my attention was taken up in recording the heart sounds, has rendered it possible for me to obtain the satisfactory combined tracings which I figure in this essay.

In conclusion, I am conscious of the difficulty in a short essay of adequately and briefly justifying the theoretical considerations which I submit, but of which I have what I consider abundant proof. Nevertheless, the facts of Case II speak for themselves, and tell us much as to the conditions which determine the appearance of this third sound of the heart.

In spite of its shortcomings, I hope that the clinical research which I have laid before you may tend to an increase of our knowledge upon this most important but difficult subject of the expansion movement of the ventricles.

Case VI. — Anæmic Dilatation of the Heart with a well-marked Third Sound

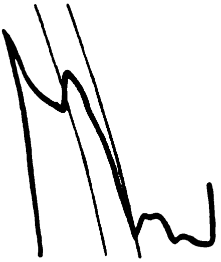


Fig. 155.—Tracing from the Apex showing a Record of the Second and Third Sounds.

A slight broad suction recoil wave is seen following the third sound.

ESSAY XIII.—ON THE CONDUCTION OF THE MITRAL SYSTOLIC MURMUR DOWN THE SPINE: ITS DIAGNOSTIC AND PROGNOSTIC VALUE[1]

THAT mitral regurgitation is characterised by a murmur audible at the apex and conducted outwards into the axilla and round to the angle of the scapula is as well known as any fact in clinical medicine ; but it is not, I think, so generally recognised that this murmur is often audible over the spines of the dorsal and lumbar vertebræ, and may even be conducted downwards into the pelvic bones. I therefore propose in this essay to draw attention to this physical sign.

The reason for the conduction of the mitral systolic murmur towards the axilla is given somewhat differently by different authors. There can, I think, be little doubt that at least two factors are at work : first, the conduction of the murmur along the ribs and chest wall, against which the apex of the heart is pressed during systole; and second, and most important, the conduction of the cardiac murmur through the layer of lung tissue which separates the left ventricle from the lateral chest wall. The greater the hypertrophy and dilatation of the left ventricle the thinner will this layer of lung become and the more easily will the murmur reach the lateral and the posterior chest wall.

CONDUCTION OF THE CARDIAC SOUNDS TO THE BACK

Neither of these reasons suffice to explain the loud conduction of this murmur down the vertebral column, which can be observed in a well-marked case of mitral regurgitation. The conduction of the mitral systolic murmur down the spine is due to the anatomical relationship of the left auricle and left ventricle to the bodies of the vertebræ.

---

[1] Read before the Queen's College Medical Society and printed in the *Birmingham Medical Review*, October 1900.

On carefully studying the conduction of the cardiac sounds and murmurs over the posterior chest wall, it can sometimes be recognised that they are especially well heard in the left mid-interscapular region over a small area about one inch or more in diameter just to the left of the spine, opposite the fourth and fifth dorsal spines. For making this observation a phonendoscope or similar powerful stethoscope is often needed.

RELATIONSHIP OF THE HEART TO THE VERTEBRÆ

The audition of the cardiac sounds over this area is, no doubt, due to the proximity of the heart to the chest wall. Under normal circumstances, the lung tissue which intervenes is sufficient to prevent the cardiac sounds from being conducted to the back. When, however, the left auricle and left ventricle are dilated, they push the posterior margins of the lungs aside and come into more or less intimate contact with the bodies of the vertebræ and the posterior chest wall. This may be easily demonstrated post-mortem in a case of dilatation of the left auricle, by distending the heart with hard paraffin prior to its removal from the body. On removing the heart after the paraffin has been allowed to cool and harden, it will not infrequently be found that there is a distinct groove on the posterior aspect of the auricle, showing where it was pressed against the bodies of the vertebræ. Since bone is a good conductor of sound, this intimate contact of the dilated auricle with the vertebral column will ensure that any sounds which may be produced in the auricle during its period of distension, will be well conducted down the spine—in just the same way as the tracheal sounds are often so well conducted by the cervical vertebræ to the back of the neck and the occiput.

These conditions occur in the case of mitral incompetence, for the regurgitating blood distends the auricle while the murmur is being produced. Further, since the axis of the left ventricle is directed somewhat backwards as well as upwards, the murmur-producing stream of blood, on escaping through the leaky valve, will impinge upon that part of the auricular wall which is in most intimate contact with the vertebræ, and thus favour its conduction through them.

Clinical Value of this Sign

We are now in a position to make some estimate of the value of this sign of which we are speaking. In the first place, we can assert that the conduction of a mitral systolic murmur to the back through the bones of the spinal column is evidence that there is a considerable amount of mitral regurgitation, and *ceteris paribus*, the extent to which the murmur is conducted downwards is a fairly reliable index of the extent of the regurgitation.

We may also consider that the loud audition of cardiac sounds in the left interscapular area and over the vertebræ, is distinctly suggestive of the presence of dilatation of the left auricle and left ventricle.

Diagnostic Value

The conduction of the cardiac sounds to the interscapular area affords us very material help in the, at times, difficult task of distinguishing between a mitral and a tricuspid murmur. I have often known the diagnosis in such a case to be satisfactorily cleared up by hearing the heart sounds pure and free from murmur in the interscapular area by means of a phonendoscope. As the left ventricle and left auricle come into closer relationship with the vertebræ than the rest of the heart, a systolic murmur, if arising in the left ventricle, is sure to be heard at the back, if the cardiac sounds can be heard there with any degree of clearness. The reverse of this does not hold true, because the mitral systolic murmur is not the only one that is conducted to the back.

The aortic systolic murmur is conducted along the line of the aorta, but it is more loudly heard over the upper part of dorsal vertebræ than over the lower, whereas the mitral systolic murmur, when heard posteriorly, is best heard near the fourth or fifth dorsal spines, and rapidly becomes fainter or disappears on passing upwards towards the higher dorsal vertebræ. Again, an aneurysm of the descending arch or thoracic aorta may give rise to a systolic murmur in the interscapular area.

TRICUSPID SYSTOLIC MURMUR

In certain cases where there is considerable enlargement of the right side of the heart, without any great corresponding enlargement of the left heart, the right ventricle and right auricle may come to be in such close relationship with the vertebræ that a tricuspid systolic murmur is conducted to the back in the interscapular area. In such cases, I have noticed that the murmur is louder on the right side of the spinal column, instead of on the left, as in the case of the mitral systolic murmur. Also there is not much likelihood of confusion, because in cases where a tricuspid murmur is conducted to the interscapular area the regurgitation is usually of so pronounced a type that there is no difficulty in recognising it in other ways.

FALLACIES

We must, of course, remember that physical signs are rarely, if ever, pathognomonic of any single condition, and in the case now being considered, causes, other than those above referred to, may lead to an increase in the loudness of the cardiac sounds or murmurs as heard posteriorly. Thus, for example, consolidation of the lung, which normally intervenes between the posterior aspect of the heart and the chest wall, may lead to an increase in the loudness of the heart sounds at the back.

Or, again, the normal cardiac sounds may be abnormally well conducted to the posterior chest wall through the interposition of a mass of new growth, as in a tumour of the posterior mediastinum. The same result may be produced in tumours of the anterior mediastinum when the heart is pressed against the vertebræ by the new growth. In this case, the probability of a tumour may be suggested by the increased loudness of the tracheal breath sound over the upper dorsal spines, which results from the pressure of the lower end of the trachea or the bronchi against the bodies of the vertebræ.

Cardio-respiratory Murmurs

Finally, I must speak of a sound which appears very closely to resemble a systolic mitral murmur, and which may be very clearly audible posteriorly. I refer to the systolic respiratory puffs which are not infrequently heard over the lower lobe of the left lung in nervous subjects when the heart is acting excitedly; or when the normal relationship of the heart to the lungs is disturbed by deformity of the chest; or by pleuro-pericardial adhesions, and similar conditions.

In the cases referred to, a systolic sound is produced which may most closely resemble a systolic intra-cardial murmur. It is a cardio-respiratory sound, due in some cases to the pressure of the heart or large vessels upon the bronchi which go to the lower lobe of the lung. One bronchus, in particular, runs close to the lateral aspect of the left ventricle, and it is, I think, quite possible that it might be slightly pressed on by a dilated and excitedly acting heart. In most cases, its true nature is shown by its variation with respiration. It is often only audible during inspiration, and is usually not heard if the breath be held. Cases are, however, by no means uncommon, where this spurious murmur continues even when the breath is held, and then the resemblance to an intra-cardial murmur is very close.

The points of diagnostic importance are—that the point of maximum intensity of this sound is usually *not* at the apex of the heart. It may be an inch or so external to the apex, or the sound may be equally well heard over a considerable area in the lower axillary region.

The anterior margin of the area over which the sound is heard often comes up to within an inch or so of the cardiac area; but the sound is not well heard over the heart itself, as would be the case if it were intra-cardial.

Posteriorly, the sound is sometimes very clearly heard; but instead of its being best heard in the mid-interscapular area, and well heard over the vertebræ, it is heard equally well over the greater part of the lower lobe, but especially below the angle of the scapula, and it is not heard over the vertebræ at all.

Prognostic Value

From what I have said, the prognostic value of the conduction of the heart sounds to the interscapular area and down the spine is evident.

If in a case of mitral regurgitation the sound is not well heard in the interscapular area, and is not conducted down the vertebræ at all, the prognosis is good, however loud the murmur may be at the apex. There cannot be much regurgitation without its showing posteriorly.

If, on the other hand, the murmur is audible all down the spine to the lumbar region, the damage to the mitral valve must be considerable, and the prognosis correspondingly grave. Simple loudness of the murmur in the interscapular area without loud conduction down the spine is suggestive of a failing and dilated left auricle.

In conclusion, I do not wish to imply by what I have said that the study of the mode of conduction of the mitral systolic murmur to the back should supplant as a method of precision those already in use—such as the careful estimation of the apex beat, the size of the heart, and the general symptoms of the case; but I am certain that it does prove an important addition to our means of estimating the amount of damage which the mitral valve has sustained in cases of mitral regurgitation.

PART IV.—SUNDRY ESSAYS BEARING UPON THE DIAGNOSIS OF HEART FAILURE

ESSAY XIV.—THE DIAGNOSTIC AND PROGNOSTIC IMPORTANCE OF PALLOR AS A SYMPTOM OF HEART DISEASE AND HEART FAILURE[1]

IN the following essay, I wish to draw attention to the diagnostic and prognostic value of pallor, which is not due to anæmia, when it occurs as a symptom of heart disease and heart failure.

But before discussing the relationship which pallor without anæmia bears to heart failure, it will be best, in order to make the subject of this paper more clear, to refer briefly to the various well-known ways in which anæmia and pallor may be associated with cardiac failure and disease.

I. The relationship of anæmia to cardiac disease may be purely accidental, as when a patient with some form of heart disease becomes anæmic through loss of blood from gastric ulcer or other cause, unconnected with the heart. To this I need refer no further.

II. Anæmia may be the cause of the cardiac failure, as is seen in the breathlessness of the ordinary anæmia and chlorosis which occurs in adolescence, and also, in part, in so-called pernicious anæmia.

In these cases of anæmia, the clinical evidence is clear that the due circulation of the anæmic blood through the lungs does—for some cause or another—necessitate the maintenance of a great increase in the blood pressure in the pulmonary artery, and, consequently, increased power on the part of the right ventricle. In proof of this, it is only necessary to point to the greatly increased loudness of the pulmonary second sound. This increase in the blood pressure in the right ventricle and pulmonary artery, does, in these cases, often exceed the resisting power of the thin part of the anterior wall of the right ventricle and the so-called conus arteriosus, leading to their giving way and becoming dilated. This dilatation, as already described in Essay I, is evidenced by increased cardiac dullness and pulsation in the third and second left interspaces and by a systolic murmur arising in the aneurysm-like dilatation of the pulmonary artery, which the raised blood pressure produces by the over-distension of its relaxed walls.

---

[1] **Read before the Birmingham Branch of the British Medical Association, March 11, 1908.**

III. Anæmia may be concomitant with the heart disease and due to the same agency.

For instance, in ulcerative endocarditis, the anæmia is the result of the toxæmia and blood destruction, which characterises the disease, and is due to the same morbid process as the endocarditis. Similarly, in systemic infection by the diplococcus rheumaticus—such as is seen in rheumatic pneumonia—both the anæmia and the cardiac damage result from the activities of the same micro-organism.

IV. Anæmia may be truly symptomatic — and result directly from the faulty condition of the heart and circulation. I need not dwell long upon this subject, as its importance is too well known to the profession, although, in practice, the value of iron in the treatment of heart disease—especially chronic valvular disease—is sometimes forgotten.

One of the most potent agents in producing anæmia in heart disease is the digestive failure, which results from the faulty circulation in the stomach and intestines, &c. It is, therefore, acknowledged that careful attention to the diet and digestive organs is a very important part of the routine treatment of heart disease. Good nutrition for the heart is in such a case of far more value than stimulants ; and the danger of over-stimulation, when the blood is of poor nutritive value, must always be borne in mind.

When present, then, anæmia must be treated energetically, for its presence is doubly damaging to the heart. Firstly, by interfering with the proper supply of food and oxygen to the heart muscle, and secondly, because the circulation of the anæmic blood through the lungs throws additional strain upon a right ventricle already fully burdened by its endeavour to supplement the faulty action of the left ventricle.

In anæmia, therefore, complicating heart disease, iron, in some form, will often prove a far more valuable restorative than the drugs which we class as cardiac tonics pure and simple.

With these preliminary remarks upon anæmia, I now come to the main subject of this communication—namely, the occurrence of pallor of the face, &c., without any marked anæmia, as a symptom of cardiac disease and cardiac failure. For the sake of brevity and for want of a better word, I wish to use the word 'pallor' in a limited and restricted sense in the remainder of this essay—namely, for paleness due to deficient circulation and not that due to a deficiency in the colouring matter of the blood, of which I have already spoken, under the term anæmia.

PALLOR IN TRANSIENT HEART WEAKNESS

Pallor, apart from anæmia, is an important sign of faulty action of the heart, and in its most extreme and simplest form is seen in an ordinary faint.

In syncope, there is no doubt as to the cause of the sudden pallor of the face, with its as sudden return of colour when the cardiac action regains its normal force.

Here, the blanching of the face may be due *simply* to deficiency of circulating blood ; but, more probably, is also of vasomotor origin, and due to a contraction of the superficial blood-vessels, whereby the maximal amount of the feebly circulating blood may be diverted from the skin to the brain.

PALLOR IN CONTINUOUS HEART WEAKNESS

In the more chronic cases of heart weakness, there may be pallor of the face, due to continuing feebleness of the circulation, which closely resembles, in its nature, the acuter pallor of cardiac syncope. But its true cause may escape recognition, and the cardiac weakness, which is really due to affection of the myocardium, may be mistaken for weakness secondary to anæmia. The pallor may be considered to be the cause of the cardiac weakness instead of its result. In such a case as this, the physical examination of the chest would help to clear up the diagnosis. If true anæmia were the cause of the weakness, there would, if the patient were young, almost certainly be some dilatation of the right ventricle upwards and to the left, and also increased loudness of the pulmonary second sound and other signs of a labouring right ventricle. If, on the other hand, the paleness of the face were a symptom of the weak heart and not its cause, there would be a diminution in the area of cardiac dullness, weakness of the cardiac sounds, and, in all probability, a high diaphragm as well. In addition, there might also be a marked diminution in the area of the liver dullness, from shrinkage of the organ, owing to emptiness of its blood-vessels.

In speaking thus, I do not mean to imply that pallor is a necessary symptom of myocardial weakness—at all events, in adult life—because, in many instances, the colour of the face may be quite good, in spite of much myocardial weakness. Pallor, nevertheless, is, when present, an important guide as to the amount of myocardial weakness present. It is, however, in valvular disease of the heart that the symptom of pallor affords us most help with regard to the diagnosis and prognosis of our cases.

One of the best recognised instances of pallor is that which occurs in aortic regurgitation. In this condition, we associate a certain patchy paleness of the face with the other signs of a severe case, and we note that the greater the degree of pallor the worse is the prognosis.

The pallor in these cases is certainly due (as we were taught at Edinburgh, by Professor Grainger Stewart, in 1881) to the failure of the heart to maintain the proper amount of blood in circulation.

To make this clear, I will go somewhat in detail into the theory of compensation in valvular disease. Compensation in aortic regurgitation depends (as was clearly expressed in 1889 by Dr. Davis in his 'Circulation of Blood through Diseased Hearts') upon two physical changes in the ventricle.

Firstly, it must enlarge so that it can hold the normal amount of blood which has to pass on into the circulation, in addition to the amount which will leak back out of the aorta into the ventricle.

Thus, if the normal charge be 3 ounces and there be a ½-ounce leakage at each beat, the ventricle must enlarge so as to hold rather more than 3½ ounces, so as to admit of the 3 ounces going forward and the ½ ounce leaking back.

And, secondly, its muscular wall must hypertrophy, so as to be able to throw this additional amount of blood into the aorta, as well as face the additional strain which the leakage causes.

If, however, the full amount of dilatation and hypertrophy, which is necessary for compensation, cannot take place, owing to the extreme amount of the regurgitation, or from some other cause, then the proper circulation cannot be carried on; for even when the nearest approach to compensation has been attained, there will still be a deficiency in the amount of blood maintained in the circulation with each beat of the heart; the arteries will be badly filled and pallor of the face will result.

In failing compensation in aortic disease, pallor of this nature is of frequent occurrence.

In the case of disease of the mitral valve, pallor, as distinct from anæmia, is not so frequent a symptom as in aortic disease, and its true importance is far more apt to be overlooked when it is present.

The explanation, in mitral regurgitation, however, is the same as in aortic regurgitation—namely, that the left ventricle is, for some reason or other, prevented from enlarging sufficiently to hold the amount of blood needed for the delivery of the normal volume into the aorta at each beat, in addition to what leaks back into the auricle.

Thus, with a leakage of 1 ounce at each beat, if the ventricle can only enlarge enough to hold 3½ ounces instead of 4 ounces, there will be a shortage of ½ ounce at each beat, and the arteries will be badly filled and pallor will result.

In such a case compensation cannot be complete, and the patient will suffer more or less from pallor due to badly filled arteries. The commonest cause of such a failure is when the ventricular wall is unduly resistant, owing to an attack of adhesive pericarditis prior to the occurrence of the mitral regurgitation. Or, in cases of endocarditis, occurring late in life, when the cardiac tissues have become less easily dilatable than they are prior to forty-five or fifty.

These cases, where pallor is due to incomplete compensation, are not very infrequent, and the cause of the pallor is very apt to be overlooked, with the result that, in all probability, too good a prognosis is given, and also the proper treatment is not adopted. For, although the treatment is apt to be tedious, much can be done for these cases by persistent care, if once the true nature of the case is diagnosed, and, in cases where adherent pericardium is the cause, cardiolysis may be indicated and give material relief.

We can all, doubtless, recall cases where we have been able to get the patient a certain distance along the road to recovery, from what was apparently a fairly simple mitral regurgitation, and then all our efforts seemed to fail to get the patient any farther.

We were, perhaps, struck by the amount of anæmia present, and felt that our efforts must be directed to cure that ; and therefore we did our best, by careful dieting and the free use of iron and other hæmatinics, to combat this symptom—but all to no purpose, the anæmia remained as obstinate as ever.

It was so evident that we did not think of making a blood count ; but had we done so, we should probably have been surprised to find that the result gave a far better percentage of corpuscles and hæmoglobin than we expected.

The careful examination of such a case would probably bring out the following facts :—

There was a loud mitral systolic murmur, conducted to the axilla and heard very clearly in the interscapular area (over the site of the left auricle) and conducted loudly down the vertebral column to the sacrum, and, perhaps, audible even over the great trochanter of the femur—suggesting a very considerable amount of regurgitation and also a considerable degree of dilatation of the left auricle—which might even be evident on percussion in the left interscapular area.

In spite of these evidences of copious regurgitation, we should be surprised to find that the apex beat was only displaced outwards and downwards to a very moderate extent, pointing to but little dilatation and hypertrophy of the left ventricle.

If the patient had not passed middle age, and especially if adolescence had not been passed, a careful search for signs of adherent pericardium in such a case as I am now describing, would, in all probability, give positive results, and there would be a suggestive amount of systolic retraction in the epigastrium or near the apex, or—what is a still more important sign—there would be evident, albeit faint, systolic retraction over the lowest ribs on the left side at the back, or laterally along the line of the insertion of the diaphragm, showing that the diaphragm was fixed to the heart by the adhesion of the pericardium and was jerked with each beat of the heart. In such a case as this, I have been able to obtain a fairly perfect cardiogram, from over the tenth rib in the mid-dorsal line, some 4 or 5 inches away from the nearest point of the cardiac impulse.

The following is a case in point :—

A girl, H. W., aged fifteen, was admitted to the General Hospital in August 1906, suffering from mitral regurgitation and stenosis with some pericarditis. After two months' treatment she had much improved, but was still very breathless on exertion and was also extremely pale. She next spent three months in a convalescent hospital, and continued to improve slowly, although the pallor did not yield to treatment at all. In February 1907, her condition was as follows: Pallor slightly less but still a marked feature of the case. Heart extremely dilated. Apex in the seventh interspace in the anterior axillary line. Well-marked mitral systolic murmur audible at the back and down the spine, also a presystolic and mid-diastolic murmur at the apex. There is marked dilatation upwards and to the left of the right ventricle, with a loud pulmonary systolic murmur, and some dilatation of the right ventricle to the right. There was systolic retraction of the epigastrium, and also systolic movement of the lower ribs on the left side—suggesting pericardial adhesion.

There was decided increase in the amount of dilatation of the right ventricle on exertion.

She was still very breathless on exertion.

She was cautioned to be very careful not to do anything that caused dyspnœa, and during the next twelve months steadily improved in every way, and, by February 1908, was so much better that she had recommenced work. The pallor had greatly lessened; the right ventricle, though still inclined to dilate on exertion, was very much smaller.

As she was only seventeen years old, the prognosis was good for a fairly useful heart, if she continued to take care.

The following is another case, where pallor was a marked feature of a case of extreme mitral regurgitation :—

E. W., a girl aged about twenty, admitted to hospital, suffering from extreme dyspnœa, marked pallor and anginal attacks—such as are commonly seen in aortic disease. These symptoms, together with a rapidly emptying pulse and extreme pulsation in the neck, made a clinical picture closely resembling that of aortic regurgitation. The pulse had not, however, the true aortic characteristics; the neck pulsation was venous and not arterial, and auscultation showed it to be a case of pure mitral regurgitation with no aortic murmur. There was, however, extreme tricuspid regurgitation.

The girl died in an anginal attack, shortly after admission. The post-mortem examination showed most marked mitral incompetence. When the ventricle was full, there was a crescentic gap ¼ inch wide by ¾ inch long. On distending the heart, the left auricle held 480 c.c. (or about 16 ounces), and the left ventricle held 110 c.c. (or nearly 4 ounces). The right auricle held 175 c.c. (or about 6 ounces), and the right ventricle, which was hindered from enlarging by adhesions of the pleura to the pericardium and to the chest wall, and also the strength of its columnæ carneæ, held only 85 c.c. (or under 3 ounces). The pericardium was adherent, but not firmly so.

In this case, the extreme amount of the regurgitation evidently exceeded the compensatory power of the left ventricle, and the difficulty was further enhanced by the pericardial adhesions hindering the enlargement of the left ventricle. The dilatation of the left auricle up to 480 c.c. was eloquent testimony as to the inefficacy of any compensation for the valvular disease.

There was abundant cause for the emptiness of the arterial system and the pallor which resulted.

A closely analogous case has been published by me in another connection :—

The girl, aged twelve, suffered from exactly similar symptoms, and marked pallor was a prominent feature of her illness. At the post-mortem, the pericardium was adherent. The left ventricle had not enlarged at all, holding only the normal 90 c.c., in spite of extreme incompetence of the mitral valve, whereas the left auricle held 164 c.c. (*vide* p. 433).

The rigidity of the heart wall, which may occur in later life, is as potent as adhesion of the pericardium in preventing compensatory dilatation of the left ventricle. When endocarditis occurs late in life, the prognosis is on this account very grave. I am indebted to Dr. J. H. Clayton for the notes of a most characteristic case of this nature, occurring in a patient under his care.

The patient was a gentleman, aged sixty-three, who, two years ago, was told by one of the leading London physicians that his heart was normal. Twelve months ago—namely, early in spring, 1907—he had an attack of illness, which was thought to be influenza, and was accompanied by a certain amount of pneumonia. Later on, he became extremely pale and began to have dyspnœa on exertion. He consulted the same physician again, who found he had a systolic mitral murmur. Later on, when Dr. Clayton saw him, there was well-marked tricuspid regurgitation with pulsation of the veins of the neck, as well as the mitral regurgitation.

As the result of treatment, the tricuspid regurgitation lessened, though the murmur persisted. But the pallor and the breathlessness remained much the same.

Iron and cardiac tonics seemed powerless, and the patient finally came to the conclusion that he was better when he did not take drugs, but simply kept quiet.

The pallor was continuously a most marked feature of the case.

This patient, as was to be expected, died suddenly in a syncopal attack. He had a slight attack of pneumonia, and, thirty-six hours after its onset and with a temperature of

101° F., he tried to get out of bed, and was found dead on the floor of his room. Such sudden heart failure is most frequent in these cases. The explanation of the case is as follows :—

This man, aged sixty-two, with rigid fibrous tissues, had an attack of endocarditis, in the course of a febrile attack (suggestive of some septic infection), and developed, as a consequence, mitral regurgitation. The inelastic and undilatable character of the cardiac tissue prevented compensatory dilatation of the left ventricle from taking place.

In thinking of such a case as this, the question arises whether we must not regard the softening of the tissues of the heart, which occurs in acute rheumatism, as a merciful provision ; for it more readily enables the heart to dilate to a degree sufficient for compensation in cases where permanent valvular incompetence occurs.

As regards the treatment of these cases of pallor, very great care must be exercised in the use of cardiac stimulants—such as strophanthus and digitalis. The forcible contraction of the ventricle is apt simply to result in further dilatation of the auricle. Nevertheless, in mitral cases, the careful use of digitalis, combined, in the later stages, with gentle exercise, holds out the greatest chance of relief.

In young patients, very considerable improvement will take place as development proceeds, if the greatest care be taken to give gentle graduated exercises (short of fatigue and of dyspnœa), and, at the same time, to maintain the general health at a high level.

In older patients, some improvement can be gained by care, but there is less probability of complete compensation ever being established. Where, in young subjects, there is certain evidence of adherent pericardium and every reason to believe in the soundness of the myocardium and in the recuperative power of the heart, the relief possible by removal of ribs from over the heart, by the operation of cardiolysis, must not be forgotten.

As regards prognosis, it is important to remember that, where marked pallor not due to anæmia is a prominent symptom in mitral or aortic regurgitation, there is very great risk of sudden death from asystole. In all cases of marked pallor associated with aortic regurgitation, the ordinary risk of sudden death is enhanced ; and where the same marked pallor is a symptom of mitral regurgitation, there is also a great risk of sudden death. If anginal attacks have already occurred in a case of mitral regurgitation with pallor, this risk is very great.

The importance of recognising the meaning of this type of pallor, from the point of view both of prognosis and treatment, is my excuse for dwelling, perhaps, at too great length upon what may be to some a familiar subject.

ESSAY XV.—SOME LEG PAINS OF CIRCULATORY ORIGIN

THE 'pains in the leg' with which I am about to deal are of circulatory origin, coming on during and after exertion, and are apt to give rise to a very considerable degree of discomfort and disability to the patient. The real cause of such pains is frequently overlooked.

I propose to discuss the following types of leg pains :—

I. PAINS DUE TO DILATATION OF THE DEEP VEINS OF THE THIGH.

II. PAINS RESULTING FROM DEFICIENT BLOOD-SUPPLY TO THE MUSCLES.

Due to either :—

1. Weak general circulation from cardiac asthenia ; or—

2. Some local interference with the blood-supply to the muscles.

(1) By arteritis obliterans (intermittent claudication, so called) ;

(2) By some obscure vasomotor abnormality. Either—

  (*a*) Arterial spasm (?).

  (*b*) Sluggish vaso-dilatation on exertion (?).

I. LEG PAINS DUE TO DILATED DEEP VEINS OF THE THIGH

Under this heading, I am dealing with a type of pain which is extremely common – especially among working-class women, old or young, and which also occurs occasionally in those higher in the social scale, but which occurs very rarely in men.

In its lesser degrees it consists simply of the aching pain usually associated with muscular weariness. In its extreme degrees it is an almost unbearable pain, which prevents the sufferer from taking active exertion or doing hard work without long periods of rest.

Sometimes the pain is mistaken for rheumatism; sometimes it is hardly felt as a pain, but as an unbearable discomfort associated with much nervous restlessness and irritability.

Varicose veins of the legs when superficial are a well-known occasional cause of pain; but it is, I think, not so widely known that dilatation of the deep veins of the thigh can be a cause of troublesome and even of incapacitating pain on walking or exertion.

The reason why this cause of pain has not obtained that degree of recognition which its frequency and its importance deserves is, I think, largely due to the fact that frequently in severe cases the pain or discomfort is either not localisable by the patient to the thigh concerned, or is apt to be mistaken for rheumatism, whereas in the less severe cases the patient does not refer specially to it—it is so constantly present—but speaks of her trouble as tiredness or weariness.

PATHOLOGY

To those who have been on the look-out for these cases there can be no doubt as to the fact that defective venous circulation is the cause of the pain, but it is not possible to feel the same certainty as to the way in which the dilatation of the veins produces the symptoms.

On anatomical grounds it seems likely that the symptoms may arise from pressure on the nerve trunks by the dilated veins, where they are confined in the same sheath of fascia. Or, again, the symptoms may be due to irritation of the sympathetic nerves which surround the blood-vessels.

A third alternative—which I think, however, can be dismissed in a few words—is that the faulty venous circulation causes a retention of waste products in the muscles and so gives rise to pain and weariness on exertion. If this were the explanation, it would bring these cases into line with those to be discussed later on. As the prime cause of this condition it must, however, be rejected in favour of one of the first two, because the pain of muscle weariness and exhaustion is, in my experience, always localisable in the muscles concerned, and is not felt as a referred or indefinite pain, as is so often the case with the severe thigh pains of which I am speaking. It is, however, quite likely that faulty nutrition takes some part in causing the aching thigh pains and weariness of which some patients complain.

One point is clear from the symptoms, and that is, that in the more troublesome cases the pain does not arise in the ordinary way, but reaches the spinal cord through channels not usually concerned with the origination and conduction of common sensation.

Fortunately, the exact pathology of the condition is, from a practical point of view, immaterial, because the treatment is the same—these cases being curable by means of gentle support applied to the thigh.

This fact, together with the frequent presence of dilated venules in the skin and a tendency, in the more severe cases, for the feet and legs to swell at the end of the day, makes it practically certain that dilatation of the thigh veins is, as I have said, the cause of this condition.

Nature of the Pain and Discomfort

1. The pain complained of may closely resemble the ordinary aching of muscular weariness, except that it comes on after an amount of walking or standing, which would not produce any discomfort in a normal limb, and the pain will be located in the thigh concerned and especially towards its anterior aspect.

The pain is usually more readily caused by standing than by walking, although in some few patients the reverse is the case, pain only coming on after such exertion as going upstairs.

It varies in degree from a simple bearable aching up to a very severe pain.

When the pain has once become severe it is usual for it to get worse very rapidly and compel the cessation of exertion. The patient will use some such language as this : 'If I once let it get bad, I find I can't stand another minute, because it makes the pain so much worse that it becomes unbearable.'

2. The pain may not be localisable to any particular part of the thigh.

Originating, as it probably does, in the nerve trunks or in sympathetic nerves, it is only appreciated as an irritation of the spinal segment or segments concerned and will be interpreted in one of the ways in which an irritation, which does not come from the ordinary sense organs, is interpreted. It may, therefore, be felt as an indefinite pain 'somewhere' about the thigh or hip, but one which the patient cannot localise or describe, coming on as an indefinite aching discomfort, and in some cases of such severity that its onset is greatly dreaded.

3. The pain may be felt as a localised 'referred pain'—a pain in some definite spot distant from the site of origin, but situated in the special skin area or areas which correspond to the special segment of the cord which is irritated by the afferent impulses coming from the thigh. Diagrams of these special skin areas, as worked out by Dr. Head and others, are now fairly common in medical literature.

4. The nervous irritation may express itself by a general disturbance of the nervous system, causing ill-defined discomfort, which is sometimes of a peculiarly distressing character. It is a sensation difficult to describe to those who have not felt it, and is always referred to by patients as being worse than actual pain. They will use such expressions as, 'If it were only a pain, I could bear it.' It is sometimes called a 'sickly aching pain.' It seems to have a more disturbing effect upon the nervous system in general than ordinary pain has, and sometimes seems to be potent in inducing that irritable weakness of the nervous system which is characteristic of the condition known as neurasthenia, just as is the case with irritation of the abdominal sympathetic nerves by nephroptosis and other displacements.

This irritability of which we are speaking sometimes expresses itself in muscular restlessness, either limited to the leg involved by the venous dilatation—as in the case of a woman aged twenty-eight, who came to see me with the complaint that she 'had the fidgets in her left leg at night and an aching pain in it which prevented her from keeping it still'—or else as that general muscular restlessness of the whole body which is so aptly described in popular parlance as 'an attack of the fidgets.' The extraordinary and distressing restlessness and inability to remain still in any one position for more than a few seconds, which characterises this condition, is apt to come on in the evening when fatigue not only tends to increase the dilatation of the veins, but also to lessen the inhibitory powers of the nervous system. While suffering from such an attack, a patient will try to describe her sensations by saying, 'Wherever I am, I cannot rest, but want to be somewhere else.'

Whenever this symptom occurs some form of irritation of the spinal cord, through other than the ordinary channels for common sensation, should be looked for.

I will now give an illustration of the more severe type of indefinite pain from this cause:—

A. C., aged about twenty-six, domestic servant, came to me complaining of neuralgia in the left hip, aggravated by exertion.

The pain was of a deep-seated character, and she could neither definitely describe it nor localise it. All she could say was that it was somewhere in the region of the great trochanter and the anterior aspect of the hip joint, and when bad it was felt down the leg as well. The pain came on with anything of the nature of over-exertion—whether by walking or using her full force in any way; she therefore dare not put full energy into any of her work. She dreaded the pain coming on, because when it once began it continued and prevented her from doing anything at all until she had had a day's rest in bed. When the pain came on it was very hard to bear and made her extremely irritable.

It was supposed that the pain had something to do with a fatty tumour of the left hip which had been removed eight years ago. She had pain before the operation, but the removal of the tumour did not cure it. Two months' rest, together with cod-liver oil, however, sufficed to cure it for a time.

The indefiniteness of the pain, its distressing and irritating character, and its relationship to exertion, all suggested the probability that it was due to dilated deep veins of the thigh.

This diagnosis was amply confirmed by the treatment, for the use of properly applied elastic webbing bandages for leg and thigh entirely relieved her, and, instead of being in constant fear of the pain, she was able to take long country walks and do hard work with a freedom from pain which she had not known for ten years.

I give a very good illustration of a case with a definitely localised referred pain, later on, when discussing the diagnosis.

Although I might multiply illustrations of the various types of pain here referred to, it is not, I think, necessary, as the cases are, as a rule, easily diagnosed when once the possibility of their occurrence is recognised ; for the condition is, as I have said, an extremely common one. It is, moreover, a cause of a great deal of misery which might very easily be prevented if its curability by the simple expedient of elastic bandaging of the thigh were more generally known.

Although the diagnosis is, as a rule, quite a simple matter, a case is occasionally met with where the true cause of the patient's pain and suffering may very easily be overlooked. The case above recorded is an instance of this. The same is true of those cases where the pain is referred to some distant spot, and the more so as one of the spots to which the pain may be referred is in the groin—a spot to which pain is referred from many different situations.

Another reason for error is that the patients may be so used to their thigh pains that they do not think of speaking of them. The following is such a case :—

A lady came to me complaining of ill-health and some neurasthenic symptoms and inability to do as much as others could without getting unduly tired. On being asked if she had any pain anywhere, she said, ' No, except indigestion ' ; but when questioned further as to whether her legs ever ached badly, she said, ' Oh yes, they always ache at night or after walking much, but then everybody's legs do.'

In the case of the lady in question, her ill-health was almost entirely due to her leg pains, and the use of properly applied elastic support to the legs and thighs not only relieved her of all her pains, but, after a few months' perseverance, they so completely restored the tone of the veins that she was able to leave them off and do a full day's shopping with less sense of tiredness than she had known for very many years. In this case the real cause of her tiredness and want of energy might very easily have been overlooked.

A very real difficulty in diagnosis will sometimes arise when the pain is simply referred to the groin ; and it may be extremely difficult to be certain of its cause, for many pains may be referred to this spot. Most difficulty is, however, likely to arise in distinguishing between pain due to colitis, or irritable colon, and pain due to dilatation of the deep veins of the thigh. I had two such cases under treatment at the same time—both in women. In both, severe pain in the left groin prevented them from walking more than a mile or so. In both there was extreme tenderness of the groin on palpation, and handling the tender spot was apt to cause a pain lasting for minutes or even hours. In neither could I make out any abdominal symptoms of colitis, or any cause for the pain other than dilatation of the deep veins of the thigh. The one case was quite cured by elastic support to the thigh, and the other was not. The latter I then treated for colitis, and she gradually improved. A year or two later, this second case had a somewhat sharp return of her pain on walking, and, knowing more of the wiles of catarrhal irritability of the colon than I did when treating her before, I examined the rectum and found that the highest palpable part was in a tender and irritable condition, and that pressure on it at once elicited the pain in the left groin of which she complained. The pain on walking was largely due to the pressure of a partially retroverted uterus upon this sensitive bowel. When the bowel was free from irritability the pressure was not enough to cause symptoms. Reposition of the uterus and treatment of the colitis at once materially relieved her.

In some cases the opposite difficulty arises, owing to pain having its origin in the colon being referred to the legs.

In such a case the patient may complain of pain felt down the backs of the thighs on walking, even going down to the toes; but careful investigation will conclusively prove it to be a referred pain of bowel origin.

Another cause of doubt may arise in those patients who only get pains in the thigh on going uphill or running upstairs. This is probably due to dilatation of the veins, but whatever its cause it has been cured in most of the cases I have seen by the use of elastic thigh-supports.

And now a word or two as to the treatment.

This is a simple matter in the case of dilatation of the deeper thigh veins. All that is needed is gentle support, sufficient to prevent over-distension of the veins, but yet insufficient to cause such pressure on the vein as to relieve its muscular walls of their proper work. Support without undue compression will strengthen the walls of the vein and enable it in a few months to do its work independently of any support, whereas undue compression of the vein will lead to a weakening of their walls from disuse and render the continuance of the support a necessity.

Although the pain arises in the thigh only, the whole of the leg must be supported. For the leg proper, nothing is better, in my experience, than the modern *crêpe* bandage. There is not the same risk of too much pressure that there is with an elastic webbing bandage or, still more, with the elastic stocking. The support of the thigh is a more difficult problem, owing to the tendency of the bandage to slip down. I think I have now solved the problem, and found a simple and easy method of giving the necessary elastic support; but it is with a sense of extreme want of originality that I remember it was only after adopting a cumbersome and time-exacting plan for fifteen or twenty years that I have within the last few years adopted the simple and easy plan I am about to describe.

The simplest device would be an elastic stocking, but the difficulty of getting one exactly the right tightness prevents its practicability. With, however, a small expenditure of time and skill, an elastic support can easily be made out of weak elastic webbing bandage. This is wound round the thigh in a simple spiral, without overlapping, and only stretched enough to give very gentle pressure—e.g. a 12-inch length ought not to be extended to more than 14 inches. When the bandage is in place, from the knee to the top of the thigh, and temporarily secured by safely pins, the patient (in nineteen cases out of twenty it is a woman) takes a needle and thread and fastens each turn of the bandage to the next above it along a line up the centre of the thigh, then she makes a similar line of stitchings up the inner and then up the outer side of the thigh, and then slips the support (as it has now become) round so as to bring the back to the front, and puts a fourth line of stitchings down what is now the front, thus making an elastic stocking for the thigh. Or if time be a consideration, the places for the four lines of fastenings can be marked by distinctive marks by pen or pencil by the doctor, and the marked bandage taken home and sewn together by the patient at her leisure. In order to fasten the bandage up, so as to prevent it slipping down, a tape must be sewn on down the front and also one down the back, with loops whereby the bandage may be suspended from the corsets, in the case of a woman, or to a belt in the case of a man, or it may be fixed to the combinations or pants respectively and drawn on or off with them. The front tape must be fastened when in the erect position, but the back tape must be fastened to the back of the corsets when the thigh is fully flexed, so that it may hold the bandage up when the patient is in the sitting posture.

The supports should be put on first thing in the morning and taken off last thing before going to bed at night.

If the elastic support thus made be regularly worn for a time, it can gradually be left off after two or three months of regular wear, being only used when extra exertion of any kind is expected. Sometimes only a few months is sufficient to cure the patient.

II. Leg Pains Due to Deficient Blood-supply

It is probably within the experience of many of us that overtired muscles are liable to ache, and, also, that a sudden strain on an overtired muscle is apt to give rise to a painful spasmodic condition—familiarly known as cramp. Some of us have experienced the sudden cramp which, towards the end of a long September day's shooting, is apt to interfere with our negotiating some awkward hedge, or impede our progress through some specially well-grown field of roots.

To state this fact in general terms, we can say that the contraction of a muscle which is taxed beyond its power of nutritional repair is painful, and that such a muscle is liable to uncontrollable painful spasm—i.e. to cramp.

Now, a muscle may be taxed beyond its power of nutritional restoration not only by excess of the work it has to do, as in the case of the long day's shooting or the long bicycle ride, but also through a deficiency in its supply of nutrition when the work required is *not* excessive, and we find that in a large class of cases muscular pain does result from defective blood-supply to the muscles.

This symptom of nutritional failure appears first in the leg muscles, and especially those of the calf, because they have to bear, in the act of walking, a heavier strain than is thrown upon any other muscle of the body in the daily routine of life, and pain in the calf in walking is a much commoner symptom of circulatory failure than would be suspected by those who have not made special inquiry into its prevalence.

The following is a good instance: A man, during a game of golf, began to feel unduly tired in consequence—as was shown by the sequel—of an attack of hæmorrhage from a gastric ulcer. On bicycling home, he found that a sense of weakness and cardiac oppression prevented him from riding up the least incline, and on walking, to push his bicycle, he had calf weariness amounting to a very distinct, though not specially severe, aching pain. By the time he reached home there was distinct faintness on exertion.

Here, although the hæmorrhage cannot have amounted to more than a pint or so and caused no discomfort or disability afterwards, it so weakened the circulation while it was in progress that it produced leg pains on continued exertion from muscular malnutrition.

Leg pains, due to deficient blood-supply, may be due :—

Firstly, to feebleness of the general circulation—as from myocardial weakness or degeneration.

Secondly, to some local interference with the blood-supply to the muscles, either :—

1. Of an organic nature, as from arteritis obliterans, causing the so-called intermittent claudication, or—
2. Due to some functional or vasomotor disturbance. Either : —
   (1) As a part of a more or less general condition of vasomotor spasm ; or—
   (2) As a result of what appears to be a sluggishness of the vaso-dilator mechanism, which is called into play on exertion for the provision of the extra supply of blood required.

(I) Leg Pains Due to Feebleness of the General Circulation

This symptom will be found sometimes a valuable guide to the presence of cardiac weakness, and especially of that type of cardiac asthenia which results from disease or weakness of the myocardium ; for aching pains in the calves of the legs on exertion is often well marked in cases where the heart is too weak muscularly to keep up the increased circulation which is necessary when all the arterioles in the muscles are dilated by exertion. That it is not more generally recognised as a symptom of myocardial weakness is, I think, due to the fact that the patient does not think of referring to it. He will speak of being too weak to walk far or too easily tired, and it will only be when he is asked to go into detail that he will volunteer the statement that *pain in his calves* prevents him from walking.

The occurrence of this pain on exertion—a definite 'intermittent claudication'—is often a useful guide as to the condition of the heart muscle.

For instance :—

Two patients present themselves before you : both complain of breathlessness on exertion and weakness of the heart. One is slightly cyanosed, has well-marked venous pulsation in the neck, a dilated heart, and slightly enlarged liver and anasarca of the ankles and legs. The second has no cyanosis ;

the face may have an apparently natural colour, there is no venous pulsation in the neck, the heart area is smaller than normal, the gastric resonance may be raised, say, to the upper border of the sixth rib or higher, and the absolute liver dullness decidedly diminished in area, probably not reaching to within one or two inches of the middle line, nor to within from half to one inch of the costal arch, and there is no dropsy.

In the former case the main cause of the failure of the circulation is mechanical and not due to failure in the nutrition or of the vitality of the cardiac muscle. It is a case of valvular disease or of failure of the heart from overstrain absolute or relative.

In the latter case it is a pure case of failure of the cardiac muscle, as a result of myocarditis, toxæmia, &c., or from failure of its blood-supply by disease of the coronary arteries or other cause.

In the former case, pain in the legs on walking will not be a marked symptom. It will be breathlessness that prevents the patient from getting about as freely as he used to do. In the latter case breathlessness will certainly prevent the patient from going up hills or stairs as he used to do, but when walking on the level it will be aching pains in his calves that will be most apt to trouble him.

Two such cases came to see me a few days before I began to write this essay. Both were men about forty-four; both were engaged in the manufacture or sale of alcohol; both had been perfectly well, so far as they knew, until a few months prior to their coming to see me; and in both the development of dyspnœa and inability to work was traceable to an overstrain. The first case was that of a publican with aortic regurgitation, dilated heart, and dropsy, who some twelve months ago strained himself helping to get some barrels into his cellar and had gradually got worse since. In the second case, one of myocardial failure, the man had first became conscious of heart weakness after having taken up work in the malting-room of a brewery at which he had worked for many years in another capacity. His new duties involved the shifting of heavy bags of malt and sugar, and at this work he gradually developed breathlessness, which finally forced him to give up work altogether.

On examination, his heart was irregular and weak, but its area was markedly diminished in size and there was no murmur. There was a slight relative increase in the size of the conus arteriosus of the right ventricle, suggestive of the fact that the primary failure was in the muscles of the left ventricle, rather than the right, and the pulmonary second sound was relatively loud.

The first case had no pains in his legs, but the second said: 'I don't have to walk long before my legs start to ache; I should not like to walk a mile—if I could go that far.' As soon as the pains came on, he was forced to stop and rest, for they increased rapidly in severity if he continued walking. This symptom is not an uncommon one, if it be looked for; but the patient, as a rule, does not seem to think it of sufficient importance to mention unless he be asked definitely. I have not as yet a long enough series of such cases to enable me to say what are the conditions which determine the presence or absence of this symptom in cases of myocardial weakness, for it does not appear to be present in all cases: sometimes the patient only complains of undue coldness of his legs on exertion. Neither have I as yet met with a case where exertion produced cramp as it does in the next class of case to be described. The absence of cramp in general circulatory weakness is probably due to the fact that the occurrence of dyspnœa will prevent the patient from overtaxing his muscles sufficiently to produce this symptom.

(II) Leg Pains Due to Local Interference with the Blood-supply to the Muscles

1. From organic disease of the arteries supplying them—intermittent claudication.

The cases of so-called intermittent claudication are of this nature. In them arteritis obliterans causes so faulty a supply of blood that the patient may only be able to walk a few hundred yards without pain, and any attempt to force the muscles to continue working in spite of the pain will bring on severe cramp in them.

These cases of intermittent claudication have received some attention of late in medical literature. The symptoms are essentially the same as those due to other forms of defective nutrition of the muscles—namely, aching pain in the calves and sometimes also in the thighs, coming on after the patient has walked for a few minutes and then passing off at once after resting a minute or two. In addition to the pain there is often a sense of contraction as if a tight band were tied round the muscle. Coldness of the legs is also a well-marked symptom and often increases as the exercise is continued. The patient gets colder with exercise instead of warmer.

A symptom which is far more common in this type of lameness than in the others here referred to, is the occurrence of cramp on exertion. So good an account of this condition is given by Dr. Byrom Bramwell, in Vol. V. No. 3 of his 'Clinical Studies,' that I shall not say more as to leg pains of this type here.

2. Muscular pains due to vasomotor disturbance.—Interference with the blood-supply, through some defect in the action of the intramuscular arteries of obscure origin and nature, is, in my experience, a more common cause of leg pains than the gross arterial disease just referred to.

The symptoms in these cases are similar in character to those just mentioned—as far as the leg pains are concerned; although the arterial supply is not as a rule poor enough to cause cramp in the muscles on exertion, as is the case where there is arteritis obliterans of the larger vessels.

In these cases there are no signs of any interference with the main arterial trunks. The pulsation in the arteries of the leg and foot is good, but, nevertheless, as soon as the patient tries to walk, severe leg pains come on, and the pain is proportional to the amount of walking done.

(1) In one class of case, of which I have seen several examples, the symptoms are extremely suggestive of vasomotor spasm. There are attacks of lividity or of pallor and coldness of the fingers and hands as in Reynaud's disease, or there may be attacks of coldness about the body as well as the limbs.

There is another symptom which is very striking, and that is the occurrence of coldness in the legs on exertion.

When the patient first starts to walk, the legs get warmer as a result of the exercise; but as soon as the legs begin to ache they begin to get cold, and the coldness increases the longer they walk. In cases of this nature, the arterial spasm—if such it be—is not confined to the legs, although it seems to be regularly induced there by the functional activity of the muscles.

These patients will complain of unpleasant cold sensations about the body in addition to the coldness of hands and feet which is so noticeable a symptom of the contracted arterioles due to an excess of uric acid and its allies in the blood.

To illustrate this better, I will give some notes of the best marked instance of this condition which I have seen.

Eleanor W., aged thirty-one, had been more or less ailing since she was fifteen years old. At twenty-four, her health broke down, and she had fainting attacks and breathlessness, also pain in the limbs and occasional cramp; also attacks of cold and shivering, in which her teeth would chatter. These last named often came on at night, and she would sometimes wake up in an attack. She had much pain and weariness in her legs on walking, and could sometimes hardly walk at all. The circulation in the hands was very bad and the fingers used often to go dead, and she suffered very much with chilblains, especially as a child. Also, as a girl of twelve to fourteen, she would sometimes have attacks of cramp and of coldness round the waist, which would take her breath away, and she used to have to go into shops and sit down because of them; but these attacks only came on in cold weather. She used to walk a great deal as a girl, but could never get warm while walking.

When her illness began at twenty-four years of age, there were marked circulatory disturbances in the hands; the finger-nails came off, and there were spots like the scurvy on them—a sort of hæmorrhagic erythematous blotches, to judge by her account. When she got better, after two years or so, the hands improved, but the leg pains on walking did not. For four or five years, she was much of an invalid, with heart weakness and leg pains and attacks of coldness about the body. She saw Sir William Broadbent, who is reported to have been puzzled by her condition, which at that time—as at present—somewhat resembled exophthalmic goitre, but with most of the cardinal symptoms missing.

She went to Edinburgh Royal Infirmary, and was under Dr. Byrom Bramwell, who gave her injections of strychnine and Weir-Mitchell treatment. When she left, she was very much better; the muscles were in splendid condition, she says, and she did more during the next twelve months than she had done for five years. Nevertheless, the leg pains remained, and the more she used her legs the more they ached.

She gradually got worse again, and the same treatment did not seem to do the same good.

As she was having much pain from dysmenorrhœa it seemed desirable that this should be relieved, and she therefore had the os uteri dilated, with good local results; but she was much upset by the anæsthetic, and when she left the hospital her legs were so bad that the pain came on after standing for even two or three minutes and got worse the longer she was on them. The pain was in the thigh as well as the calves, and she described the sensation as that of a tight bandage round the thick part of the thigh, squeezing it painfully and stopping the circulation.

She also got pains in the biceps and forearm muscles after continuous work such as sewing. She also had attacks of coldness of the hands and arms accompanied by much numbness, and these attacks were often followed by a sense of great heat accompanied by flushing of the hands and aching pain—similar to the so-called 'hot ache' which children experience after their hands have got unduly cold in winter.

In addition to these symptoms the pulse was 120 to 130, but without any dilatation of the heart or other definite abnormality of thoracic or abdominal organs. Some tremor of the hands also occurred and occasional sense of throbbing in the neck with a little choking sensation, but no other symptom of Graves' disease.

For the last year or two she has been very slowly improving, but only able to take very little muscular exertion of any sort without pain; she also gets frequent attacks of coldness of her hands and sometimes in the body.

The outstanding feature of her illness is pain in the legs on the slightest exertion—even walking a few times across the room will bring it on. This patient was further improved by brisk exercises of short duration—such as skipping—and by taking pituitary extract.

The following is a very different case from the last, for it occurred in an apparently healthy working man instead of in a neurasthenic young woman.

The patient, a night-watchman, complained of pain in his legs coming on in the course of his rounds and causing him to stop and rest. It used to occur in both legs, but at the time of his admission to the hospital it was mainly limited to the left leg and thigh. When the pain comes on the whole leg and thigh go cold and numb. The pain goes completely away when he rests, as does also the coldness, which gradually passes away from above downwards.

There was no sign of any disease in the arteries of the legs, and the blood pressure was good. There was some lividity at times of the hands, and they were of a deep red colour with very slow capillary reflux.

(2) Pains due to defective vaso-dilatation.—In what will, I think, prove to be another group of cases, of which I have seen one or two instances, the disturbance is limited to the legs, and there are no signs of any general vaso-motor spasm; but the condition seems to consist in some persistent interference with the supply of the blood to the muscles, or the removal of waste products from them, leading to premature fatigue. I cannot say whether this change consists of vaso-motor spasm of the muscular arterioles or sluggishness of the vaso-dilator mechanism interfering with the proper dilatation of the vessels during muscular activity—or whether, on the other hand, there is actual arteritis or pathological rigidity of the vessels; or, further, the defect is due to some interference with the removal of the lymph from the muscles.

Certainly, in some cases, the cause is of a removable nature, as is shown in the case subsequently to be narrated.

In this third group of cases, where there is no evidence of arterial spasm, the most likely explanation is a sluggishness of the controlling mechanism when the muscular arterioles are dilated in response to the call for the increased blood-supply to which muscular exertion gives rise.

A kind of ‘habit’ paresis, similar to such conditions as hysterical aphonia and many other functional errors of the nervous system.

The following most interesting case appears to be one of this nature :—

As the sufferer is a doctor, who has kindly sent me notes of his own case, it has additional scientific value. His experience is as follows :—

In November 1907, he began to have pain in his legs and to feel unduly tired after his day's work, which, while suggestive of rheumatism, did not yield to anti-rheumatic remedies.

They gradually got worse, and were accompanied by sensations of pricking in the legs and calves, also 'pins and needles' and numbness ; also there were localised sensations of heat in the legs and a distinct 'girdle' sensation around the upper part of the calf. These sensations came on after walking half a mile or so, and would soon become quite unbearable if walking were persisted in ; also there was such weakness that he felt his legs would give way under him. Standing or sitting for a minute or two would quickly relieve him and allow of his going on without discomfort.

He remained in this condition all through the winter months and had carefully to avoid much walking. During the attacks of pain and numbness there was never any coldness, pallor, or congestion of the skin.

In April 1908, he consulted Dr. Byrom Bramwell in Edinburgh, who found that there was no noticeable disease or narrowing of the main arteries of the legs, and, as he was in the habit of doing much of his work on a motor bicycle—to the extent of some 100 miles or so per week—he advised the discontinuance of this and of ordinary bicycling, together with more walking. He endeavoured to follow this advice as far as he could, but was not able to trace any connection between the periods of aggravation and remission of his leg pains and the use or non-use of his motor bicycle. He continued much the same till June, being quite unable to walk as much as a mile without resting to allow the pains to pass off.

In June, he tried a game of tennis and played for three hours without any discomfort. He found no harm resulted from the exercise, and, on the contrary, there was decided improvement in his powers of walking. Since then, he has played tennis frequently and has remained very free from attacks or pain, although he has had many slight ones ; and in August, he once walked eight miles without any discomfort. As regards the causation of his attack, he thinks the vibration of the motor bicycle had much to do with it, and has noticed that even after driving a motor-car he has had a slight attack of the pain.

The pathology of this case is clear in so far that it shows the attacks of pain to have been due to some form of defective nutrition of the muscles.

In this case, the sudden violent movements necessary in playing tennis so increased the nutritive interchange of blood or lymph as to leave the circulation in the muscles permanently improved.

Whether the defect were in the vaso-motor nerves, the arterioles, venules, or lymphatics, either the sudden powerful call for more blood, or else the violent muscular contractions broke down the habit of quiet and ineffective circulation which had previously been established.

This curative action of powerful contraction also raises the question whether the attack of cramp which occurs in tired muscles is not also a curative effort on nature's part. The powerful contraction squeezes out the waste products that have been slowly accumulating and enables the muscles once more to do their work effectively. Certain it is that, after a violent attack of cramp which at the time seemed likely to deprive me of the last hour of a day's shooting, I have sometimes been surprised to find that I could go on walking more freely and could finish my day with no more return of cramp.

Also I remember a twenty-mile bicycle ride after a long day's fishing, where from about the fifth to the eighth mile I had cramp in apparently every muscle of both legs, but was able to ride the last ten or twelve miles with perfect freedom and enjoyment, in spite of much of the road being uphill.

Such cases as these just referred to, open up a large field for research and suggest the probability that the vaso-motor system is dependent, for its proper functions, upon some internal secretion or other special substance, and that when this is deficient in amount the vaso-motor functions are not properly performed. The last case seems to suggest the probability that, as with other functions of the nervous system, a temporary failure of function may be perpetuated as a 'habit paralysis' or a 'habit spasm,' just as is the case with hysterical aphonia and other forms of habit paralysis which can be cured by re-education, or the habit spasms which we often meet with—of which some cases of wryneck and writer's cramp are common examples. It seems quite certain that the sudden and powerful call for more blood to the muscles which resulted from the game of tennis, broke down the habit of sluggish vaso-motor response to the ordinary calls for blood which walking produced, and so cured his intermittent claudication.

I have met other cases where some such sluggishness of the vaso-dilator mechanism appeared to be the cause of the patient's symptoms, but I cannot enter upon this larger subject within the scope of this paper, and I throw out this suggestion in the hope that the attention of others may be directed to the possibility of such a condition.

I have at the present time under my care a case of troublesome neurasthenia, where the symptoms seem explicable on the supposition that the arterial blood supply is not adequately increased by vaso-dilatation when there is a call for increased activity of the brain.

I must not leave this subject without referring to a class of case which is sometimes difficult to differentiate from those cases of muscular pain from malnutrition.

I refer to those cases where the muscles are painful on exertion, but where tenderness on pressure apart from exertion is an extremely marked symptom.

These cases were first brought under my notice by a little book by Dr. Rabagliati of Bradford, very many years ago. In them the muscular tenderness and painfulness suggests some form of myositis or unusual rheumatic affection, but this theory is negatived, I think, by the fact that the condition is curable, as Dr. Rabagliati pointed out, by forcible voluntary movements, especially if combined with firm pressure over the muscles—an exercise calculated greatly to improve their nutrition.

It may be that here too we are dealing with a vasomotor failure; but the fact that the muscles are in some cases extremely tender when at rest seems to negative the possibility that malnutrition alone is the cause. It is of interest to note that this condition of extreme muscular tenderness is sometimes found in patients suffering from muscular hardening, tenderness and pain in the colon; and it is far from improbable that the condition is really due to the appearance in the voluntary muscles of a condition of tonic expansion similar to that which is giving rise to the intestinal symptoms (*vide* 'Pain in Colitis,' *British Medical Journal*, June 1909).

I have seen several cases when the muscular tenderness, curable by exercises, involved the abdominal muscles, and two where it involved most of the muscles of the body, but especially those of the abdomen and legs.

As regards the treatment of the last groups of cases referred to, the important indication is improvement of the nerve tone, and massage and galvanism with full feeding are valuable. It is also very important to look out for and combat any possible cause of neurasthenia which may be present. Also it must not be forgotten that the case may prove one of the simple ones which can be cured by violent muscular exercise, and such an exercise as the fashionable one of skipping is admirably adapted for such cases.

I feel some apology is needed for bringing forward these last-named subjects in such an incomplete form, but my doing so will, I hope, lead to the publication of other cases which may give fresh light as to the cause and treatment of these as yet somewhat problematical complaints.

ESSAY XVI.—ON THE VALUE OF REVERBERATION IN THE DIAGNOSIS, AND OF GASTRIC MASSAGE IN THE TREATMENT, OF ATONIC STATES OF THE STOMACH[1]

REVERBERATION IN GASTRIC DIAGNOSIS

ALTHOUGH we have many means at our disposal for the recognition of atonic states of the stomach, any addition to our diagnostic armamentarium will doubtless prove acceptable. This, therefore, is my excuse for this short essay.

I have for some time used, in abdominal diagnosis, a modification of the old-fashioned 'bell-sound' method of recognising large cavities in the lung, or the presence of pneumo-thorax. As commonly used, a coin pressed on the skin is struck by another coin, and the metallic reverberation of the sound is audible over the whole of the cavity under examination. In the modification referred to, instead of the clinking of coins, reverberations are set up in the cavity to be examined by scratching with the finger-nail a roughened rod, which is firmly pressed down on to the skin overlying it.

When listening with a stethoscope over the cavity itself the sound has a distinctive metallic character, and when the stethoscope is not over the cavity the scratching sound only is heard.

The area over which the scratching sound has this distinctive reverberating character defines, with great accuracy, the area occupied by the subjacent air-containing viscus, and the method is often of distinct assistance where such an air-containing organ as the stomach or the distended colon cannot be accurately mapped out by ordinary percussion. It is of distinct value where the pitch of the colon or intestinal note happens to be so similar to that of the stomach note that percussion unaided gives an uncertain result. There are two ways in which reverberation can be practised, and sometimes one and sometimes the other gives the best results. Supposing that the area occupied by the stomach is to be defined, the roughened rod is pressed down firmly upon some spot in the epigastrium where tympanitic gastric resonance is well marked, and the area over which the metallic reverberation is audible is carefully noted by the stethoscope. The alternative proceeding is for the stethoscope to be always on the same spot, over the stomach, and the reverberator moved about. When pressed over the gastric area, metallic reverberations will be heard; when not over it, they will not be heard. Of the two methods the former is, perhaps, the most convenient, if a binaural stethoscope is used, as it can be more easily moved about than the reverberator. If, however, a wooden stethoscope be used, the second method, where the reverberator is moved about, would prove the most convenient. As thus employed, the method simply shows the area occupied by the stomach, and supplements ordinary percussion.

---

[1] See *Birmingham Medical Review*, 1908.

It can, however, be made to give useful evidence as to the amount of muscular tone present in the stomach. We have but few direct methods of ascertaining this, unless the stomach wall be contracted enough to be made out by palpation (as is sometimes the case), or unless there be sufficient muscular hypertrophy for the waves of peristalsis to be visible—as in pyloric stenosis, &c. If both the stethoscope and the reverberator be over the stomach, and the reverberator be continuously scratched, and with an unvarying degree of force, the pitch of the note will often be found to vary from to time time, alternately rising and falling. The rise in the pitch of the note is due to an increase in the tension of the stomach walls and of the gas within it, owing to muscular contraction or other cause, and a fall in the note being, conversely, due to lowering of the tension by muscular relaxation. Thus, by the presence or absence of this change in the pitch of the note it is possible to say whether a somewhat enlarged or overfull stomach is due to a spasmodic contraction of the pylorus with normal or even increased tone of the stomach muscles, or whether, on the other hand, it is due, not to hindrance to the escape of the gastric contents, but to a defect in its expulsive power owing to muscular atony. In a flabby atonic stomach there would be little, if any, change in the pitch of the reverberation note. With a little experience, it is sometimes possible to say at once, on hearing the reverberation sound, 'This stomach is not atonic, the reverberation note has a high pitch.'

The value of reverberation in gastric diagnosis was evidenced by a case recently under my care in the General Hospital. The patient was emaciated and there was something suggestive of Addison's disease about her. On palpation of the epigastrium a gurgling was occasionally felt, as when the stomach empties itself through a slightly narrowed pylorus. On testing with the reverberator, however, although the gurgling could be distinctly heard during the examination, the pitch of the gastric reverberation note remained unchanged, and of a fairly low tone. This showed that the gurgling was not of gastric origin. On placing the reverberator over the situation of the transverse colon, however, and listening over it, it was found that there was a marked change in the pitch of the note, which gradually ascended till the gurgling took place, and then dropped again. On further investigation it was found that on placing the reverberator over the colon to the left of the middle line, and listening near the hepatic flexure (or vice versa) the reverberation note could occasionally be heard from the ascending to the transverse colon and occasionally not, and that the period of its audibility corresponded to the periodicity of the occurrence of the gurgling; at the time of examination about once every minute or two. This showed conclusively that there were peristaltic waves in the transverse colon—that the colon was sometimes unduly distended, allowing the reverberation note to be audible throughout the upper part of the ascending colon and the whole of the transverse colon, and that at times this continuity in its lumen was broken by muscular contraction in the region of the hepatic flexure.

On testing the descending colon it was found that the reverberation note was never obtainable between the transverse and the descending colon, showing that there was some interference with the lumen of the gut in the neighbourhood of the splenic flexure.

As the reverberation note seemed to be always obtainable throughout the transverse colon, the latter was evidently too dilated to be obliterated by the muscular contractions, and therefore, the stricture in the region of the splenic flexure could not be a very recent one or a very slight one. The presence of a partial stricture of the colon near the splenic flexure was also suggested by the fact that the sigmoid flexure could be felt to be empty and of normal muscular tone. The cæcum and ascending colon were not distinguishable by palpation.

On palpation the gurgling could sometimes be felt, and its situation seemed to be too much to the left to be due to a contracted pylorus; nevertheless, without the aid of reverberation the diagnosis could not have been cleared up as it was. There was no tumour to be felt, and nothing abnormal was palpable in the left hypochondrium except in the slight gurgling already mentioned, which occurred every minute or so. The stricture was not by any means a complete one, for there was not any serious difficulty in getting the bowels open.

Considering the cachectic condition of the woman the recognition of this interference with the action of the colon made the diagnosis of cancer involving or originating in the splenic flexure of the colon a fairly certain one.

I have recently had another case very similar to the one just recorded, but in neither case has there been a post-mortem examination to confirm the diagnosis.

Although such cases as these are by no means of frequent occurrence, they suffice to prove the value of reverberation as a method of diagnosis, and in ordinary cases of gastric derangement the medical practitioner who tries it will, I am sure, find it a useful adjunct to other methods. It does not need any elaborate apparatus. For many years, I used a small bone rod some one and a half inches long, with a roughened top which I could scratch ; but of late years I have used a much simpler implement, and one which is probably in the hands of most medical men : I refer to the small five-shilling stylographic pen. When closed, it is under four inches long and has a roughened ring round it just below the cap to facilitate the unscrewing of the writing-point. If the pen be held with the lower end in the palm of the hand and the cap pressed against the wall of the chest or abdomen, this roughened ring comes at just the right level to be scratched by the finger-nail of the first or second finger.

It is a most convenient form of reverberator, and obviates the necessity of adding another to the necessary instruments of precision which the doctor has to carry with him.

VALUE OF MASSAGE IN THE TREATMENT OF ATONIC STATES OF THE STOMACH

In a very considerable proportion of the simpler cases of indigestion which we have to treat, an atonic state of the stomach plays an important part. Catarrhal conditions of the stomach, and lowered vitality in general, are extremely apt to lead to a deficiency in the hydrochloric acid of the gastric juice, and also to a deficiency in the muscular power of the stomach. These two conditions both bring about delay in the emptying of the stomach, and cause further interference with its functions, not only by depriving the stomach of its proper rest between meals and at night, but also because the remains of a preceding meal will hamper the digestion of the succeeding one. The usual remedy for such a condition is the familiar acid and nux vomica, with or without pepsin, after meals; but a very valuable adjunct to this treatment is a form of massage calculated to assist the enfeebled muscular action of the stomach, and so prevent the damage done to the mucous membrane by the undue retention of its contents.

Before attempting to devise a means of helping the muscular action of the stomach by massage, we must study carefully its normal muscular movements. This can, fortunately, be done clinically, for its peristaltic movements are not infrequently visible in cases where there is some pyloric obstruction with some dilatation of the stomach. In such a case the passage of the wave of peristalsis is sometimes easily observable and its characters can be noticed.

The character of the peristaltic movements of the stomach is as follows:—

1. A peristaltic wave consists of a single narrow constriction of the stomach, which commences near the cardia

and passes down towards the pylorus, keeping always transverse to the diameter of the stomach ; or, to express it otherwise, the depression due to the contraction is along the line of a radius of the circle of which the greater curvature is the circumference.

2. Further, the waves occur singly and not in groups and a second wave does not begin to travel down across the fundus until after the preceding one has reached the pylorus.

3. As to rate: The rate of the wave is not quite uniform. It commences rather more slowly, and quickens slightly as it approaches the pylorus. This quickening commences to be distinctly appreciable over the one-third or one-fourth of the stomach at its pyloric end. The wave also gains in force, and is more distinctly visible over the pyloric end of the stomach than over the fundus.

Owing to the distinctness of the waves it is quite easy to time them. In an active stomach it will be found that they follow each other at intervals of about twenty seconds over any particular part of the stomach, and, as they follow one another without any interval, their periodicity may be put down as about three per minute. In a less active stomach, half a minute may be found to elapse between succeeding waves, but three per minute may be taken as the normal rate.

This periodic contraction may also be estimated by the palpation of the pylorus, and often constitutes a valuable means of distinguishing the pylorus from a nodule of new growth, or an enlarged gland. In the case of the pylorus its hardness will be found to vary periodically.

The knowledge of these facts makes it clear that movements of the hand at the rate usually adopted in general massage will not be calculated to aid the gastric peristalsis, but will, on the other hand, be apt, in the feeble-acting atonic stomach, to break up the peristaltic waves and hinder rather than help the stomach in its efforts to empty itself.

In gastric massage, effort must be made to help the stomach in two directions. Firstly, the endeavour to diminish the size of the enlarged and flabby organ so as to give the weakened and extended muscles more purchase in their effort to constrict its lumen ; and, secondly, by means of slow rhythmically varying pressure to simulate the action of the peristaltic waves, and thus accentuate the stomach's own efforts to force its contents through the pylorus.

The first indication—that of diminishing the size of the stomach—can be met by endeavouring to support the lower border of the stomach by the hand and push it upwards. To do this most effectively the patient must lie on a couch with the head and shoulders slightly raised, and with the abdominal muscles relaxed. Also, for gastric massage, the patient ought to be slightly turned towards the left side so that gravity may draw the stomach slightly away to the left and thus bring the axis of its pyloric end into line with the axis of the pylorus and first part of the duodenum. In an atonic stomach, such as we are speaking of, if the patient were to lie on the left side, the stomach would be apt to fall away rather to the right side, and a partial kink would tend to be produced thereby in the region of the pylorus.

To support the stomach most effectively it is necessary to use the right hand and to stand on the right side of the patient, so that the hollow of the right hand may coincide with the curve of the lower border of the stomach, and also so that general uniform pressure may be applied over the stomach in the endeavour to keep it pressed back under the costal arch and towards the normal position of the pylorus. The ulnar border of the hand must therefore be gently placed on the abdomen about the level of the stomach's lower border and the endeavour made to gather it up into the hollow of the hand by means of gentle pressure combined with a lifting and shaking movement.

When this is done, gentle continuous pressure is exerted by the hand for some twenty seconds or so, and then the pressure is relaxed and the hand reapplied as before, and pressure continued for another third of a minute. This must be continued for ten minutes or so. This simple type of intermittent pressure massage is decidedly effective in aiding the stomach to empty itself, and proof of this may be found in the fact that after applying it to an atonic stomach, which still contains food, some three or four hours after a meal, it will be found to empty itself very considerably if not entirely in the course of a comparatively few minutes' treatment.

Sometimes gurgling due to the passage of its contents through the pylorus will be felt within a minute or two of commencing to press the stomach.

The advantage of this type of massage is that it can be easily learned by the patient, and to be really useful this is essential, for the massage ought to be done half an hour or so before meal-times, and for twenty minutes or so before going to bed at night, so as to lessen the risk of food remaining in the stomach all night. This type of massage is extremely beneficial to the patient, although of less direct value to the medical practitioner, as it enables the patient to cure himself somewhat rapidly after he has once been taught how to do it.

This pressure massage is rendered more efficacious if, while the hand is being pressed over the gastric area, rhythmically varying pressure is exerted so as to simulate the passage of a peristaltic wave. Thus, to commence with, the finger-tips are somewhat more firmly pressed into the abdomen over the fundus of the stomach than the rest of the hand, then, after a few seconds, pressure is made specially with the centres of the fingers and later on with the digital border of the palm. Then the hand may be shifted a little so that the ball of the thumb comes to be over the situation of the pylorus, and for the last eight or ten seconds pressure is specially made by the ulnar and digital border of the palm, so as to press the gastric contents towards the pylorus, just as the final, more powerful effort of the normal persistaltic wave does.

After twenty seconds, the same manipulation is practised again; the stomach is afresh gathered up, as it were, into the hollow of the hand and pressure begun by the finger-tips just under the costal arch over the fundus, and then slowly the stomach is pressed on nearer and nearer to the pylorus, until during the last one-third or one-fourth of the twenty-seconds' period the pyloric end of the stomach mainly is pressed on and in a direction towards the pyloric orifice.

As will be gathered from this description, the hand must be flexible and well trained in order to exert a varying pressure by first one and then another part of it without relieving altogether the pressure of the rest of the hand. It would be of little value for the pyloric end of an atonic stomach only to be pressed on, for the contents would simply be pressed into the fundus. If however, general pressure can be exerted over the whole of the stomach with, at the same time, extra pressure localised over the pyloric end, the effect is very considerable. When skilfully applied this varying pressure massage is, in my experience, a potent means of emptying an atonic stomach. In the case of an educated patient this type of massage can also sometimes be taught, but the 'horny hand' of the son or daughter of toil is incapable of the differential movements necessary to produce a regularly varying degree of pressure on one part of the stomach during the maintenance of a general pressure over the whole organ. In cases, however, where the patient has been able to learn this more complicated form of pressure massage, I have often been astonished at the rapidity of the improvement in the gastric tone, and the patient has been most pleased at the result, and especially at the increase in his comfort at night, due to his being able to get the stomach to empty itself prior to his going to sleep.

It is the efficacy of this form of massage which induces me to dwell at such length upon this subject.

ESSAY XVII.—A CASE OF ARHYTHMIA WHICH THROWS NEW LIGHT UPON THE VENOUS PULSE AND THE ACTION OF THE AURICLES[1]

A FEW words must be said by way of introduction to this essay.

In the first place the writer does not expect that this essay will prove of interest to any but those who are making a special study of the heart by means of graphic records, and, moreover, to many of them his interpretations will be so contrary to the usually accepted ones that few will probably care to make the mental exertion of trying to follow them.

Nevertheless, this essay forms an important part of the message which the writer desires to make public.

Also this essay may be criticised upon the ground that the tracings are not sufficiently important to be worth so much study. In answer to this the writer would say that he has given this series of tracings as typical, and that the methods of interpretation here given are applicable to other types of tracing.

Fault may also be found with these tracings upon the ground that the cardiograms here given are different from those usually obtained by the Mackenzie polygraph, and their accuracy may on that account be thought to be open to doubt.

The answer to this criticism is that they *are* different, because they are taken by a different apparatus from that usually supplied with the Mackenzie polygraph. Whatever may be the advantages of the cup for the record of the cardiac movement it has the disadvantage that it gives only a generalisation as to the movements of the heart.

In the apparatus used by the writer, the principle of Marey's sphygmograph and Galabin's excellent cardiograph have been adapted to the Mackenzie polygraph. In this way the movement of a very small area of the heart, as transmitted through the chest wall, is recorded and magnified by mechanical means before being conducted by air to the writing tambours.

---

[1] Subject of a communication prepared for the Dublin meeting of the Association of Physicians, 1909.

In the opinion of the writer, the true relationship between cardiac activity and the venous pulse can only be adequately studied by some such apparatus.

This essay may also be thought to be needlessly full of detail. But as is pointed out in its course, an explanation of a cardiogram cannot be accepted as accurate unless it suffices to explain adequately the main features of the tracing.

In the present instance there are sometimes as many as sixteen or eighteen distinct waves associated with each cardiac cycle, and the adequate explanation of these cannot be expected to be very brief—more especially as the explanation can, in many cases, only be arrived at by comparison with other tracings which in their turn need to be explained.

The explanation of the tracings here given is, in some instances, only tentative ; but the writer feels confident that the tracings will be adequate to convince anyone who has the patience to think them out along the lines suggested in this essay.

In order that the accuracy of the correlation of the various tracings with one another may be tested, the commencement or ending of the tracing is always given when possible. When this is not done, as in the case of some of the photographic enlargements, the original tracing from which the enlargement was made is given in order that the corresponding points in the venous and ventricular portions may be measured by any reader.

SUMMARY OF THE ARGUMENT[1]

Careful study of the graphic records of the case here described seems clearly to prove the following propositions :—

1. The ventricles of the heart fill themselves by their own expansion.

2. So far as sensitiveness to mechanical stimulation is

---

[1] For summary of facts and theories here brought forward, see p. 523.

concerned there is for the ventricles no non-excitable 'latent period.'

3. The ventricles can be stimulated to contract immediately after the normal systole, by over-distension—the result of their own expansile act—thus causing an extra systole.

4. There is for the AURICLES no 'latent' or non-excitable period, so far at all events as mechanical stimulation by distension is concerned. A definite contraction can be caused shortly after the auricular systole by the inrush of blood regurgitating from the ventricle.

5. There is no true relaxation or passivity of the muscular walls of the auricle. They remain in a state of tonic activity throughout the diastole.

6. The walls of the AURICLE are, throughout the diastole, SENSITIVE to any sudden changes of pressure within it, and respond by increase of tonic activity or by contraction, to any force tending to distend it, or to any relief of pressure within it.

7. The GREAT VEINS, by means of their tonic activity, maintain the blood pressure within them at the same level as that within the auricle and ventricle (during their relaxation phase), and at such a height that the blood in the right side of the heart and veins acts as one continuous column of liquid within firm walls, the conduction of impulses being thus rendered practically instantaneous.

8. The walls of the GREAT VEINS are SENSITIVE to sudden variations in the pressure within them, and respond by increased tone or by a definite act of contraction to stimulation by sudden distension. Such contraction may be observable in venous tracings if the distensible force be not great enough to overpower the muscles of the veins, or so prolonged that the stimulus to contract loses its effect before the pressure is sufficiently relaxed to admit of the contraction emptying the vein. The vein may also be stimulated to contract by a sudden decrease in the intravenous pressure.

INTRODUCTORY

During recent years a great stimulus has been given to the study of the heart by means of graphic methods, not only by the writings of Sir J. Mackenzie, and others, but also far more by the extremely practical and convenient polygraph which we owe to him.

The clinical work has, however, been largely along one line—namely, the study of disorders in the conductivity of stimuli through the heart muscle, and many records are being published of venous tracings which show, or seem to show, auricular contractions which are not transmitted to the ventricles and which are apt to be taken as evidence of a greater or less degree of heart-block in the patients concerned.

One object of this paper is to draw attention to a case which seems to show clearly that the auricles and ventricles are both sensitive to mechanical stimulation and that extrasystoles may be explicable in this way quite apart from faults in conductivity.

Now the interpretation of graphic records of the heart or veins is by no means as simple as it appears; and I have not infrequently seen, in the medical journals, venous tracings interpreted in a manner which *may* have been accurate, but of whose accuracy the tracings given afforded no proof. The importance of a mistake in interpretation may be very great, because the presence of extra systoles of the auricle is apt to be taken as a sign of some serious defect in the conductivity of impulses through the heart, and suggests a bad prognosis.

Now waves in the neck veins may occur which closely resemble auricular pulsations, but which are not of auricular origin, and there may be extra systoles of the auricle, as this case seems to show, which are the result of mechanical stimulation and whose occurrence does not indicate any abnormality of conductivity.

Therefore, it is necessary to compare the venous tracing with a carefully taken apical one before a reliable interpretation of it can be made and before it can be asserted that the auricles are acting independently of the ventricles, or that anything of the nature of delayed conductivity is present. But, unfortunately, interpretation of a cardiogram, as is the case with other clinical problems, closely resembles the interpretation of a sentence of a foreign language: you cannot be certain of its meaning till you know the meaning of every word. A word left untranslated may be the 'not' or the 'never' which entirely reverses the whole meaning of the sentence. An illustration of this is given at p. 519.

Therefore, before I can use my tracing as evidence, I must show that my interpretation holds good for every wave and oscillation in it, and as there are sometimes sixteen distinct waves for one beat of the heart the task of interpretation must put some strain upon the patience of the reader.

But before going into the cardiographic evidence, I must describe the case in its main bearings.

The patient was a young man aged twenty-one years, a gardener by occupation. There had been a gradual onset of shortness of breath for the last year or so, combined with some pain in the left shoulder when working with the left hand. For the last one or two months he had had occasional attacks of faintness and sudden feeling of weakness, causing him to stop work and sit down if they came on by day. More usually, however, the attacks came on at night, would sometimes last an hour, and were sometimes accompanied by vertigo—a sense of objects turning round him; also he occasionally had stabbing pain in the left side of the chest on exertion—such as lifting a heavy weight. He has not been able to work for three months. He had leg pains on exertion—e.g. after walking a mile or so a sensation of weakness in his legs would come on, accompanied by aching pain in the calves, which lasted for an hour or so after ceasing to work. His feet were always cold, and, though walking would warm them for a time, they would go cold again, especially when the pain came on.

There was evidence of weak cardiac action in the fact that the gastric resonance was an interspace too high, that the liver dullness was small, the dullness of its left lobe being absent, and that of its right lobe not reaching to the costal arch. Although the man was well nourished and healthy looking, it was quite evident from his symptoms and the condition of his liver and diaphragm that the heart was not able to keep the proper amount of blood in circulation.

On listening to the heart there was no murmur, but considerable irregularity, consisting in coupled beats—namely, a normal one closely followed by a short imperfect one, which the cardiograph showed to be an extra ventricular systole. Although the extra systole alternated—as a rule with the normal beat—it occasionally occurred less frequently, i.e. once in three or four beats.

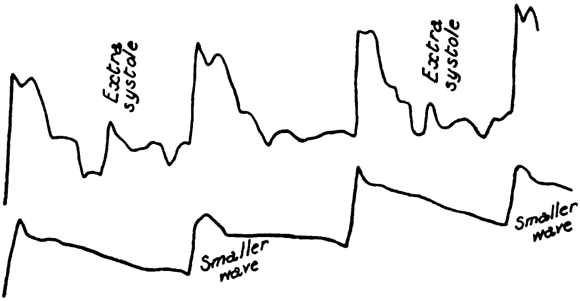


FIG. 156.—TRACINGS FROM THE APEX AND THE CAROTID ARTERY, SHOWING THAT THE EXTRA SYSTOLE CAUSES NO PULSE WAVE IN THE ARTERY AND THAT THE WAVE FOLLOWING AN EXTRA SYSTOLE IS SMALLER THAN NORMAL.

This is copied from fig. 1, Plate IX, and is not drawn to scale.)

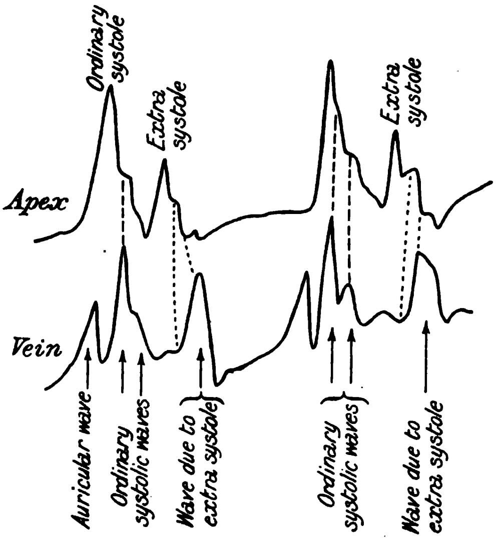


FIG. 157.—COPY OF A TRACING SHOWING A WAVE IN THE JUGULAR VEIN WHICH RESULTS FROM THE EXTRA SYSTOLE.

On taking simultaneous tracings of the apex beat and of the carotid artery and of the apex and external jugular vein the reason for the cardiac inefficiency was apparent, for the extra systole did not as a rule succeed in forcing blood into the aorta, there being no carotid pulse wave corresponding to it, whereas there was a distinct wave in the veins of the neck. Moreover, when the extra systole occurred less often than with alternate beats, it was evident that the carotid pulse wave succeeding the extra systolic was smaller and less effective than that due to the other ventricular contractions (*vide* figs. 156 and 157).

The extra systole, therefore, not only weakened the heart in so far as it was a useless contraction, but also it interfered with the amount of blood sent forward by the succeeding normal beat.

CAUSE OF THE EXTRA SYSTOLE

The extra systole, as has been said, occurred immediately after the preceding normal contraction. On studying (in the cardiogram) its relationship to the preceding beat, it is seen that it occurs immediately after the return of the systolic rise to the normal level. This point in the cardiogram marks the commencement of the phase of ventricular relaxation and the termination of the short period (one-tenth second or so) of active expansion, during which the ventricle expands with hardened wall and fills itself by its own aspiration. In the accompanying cardiogram the position of the second sound has been marked by an electric signal (*vide* fig. 158). Its position

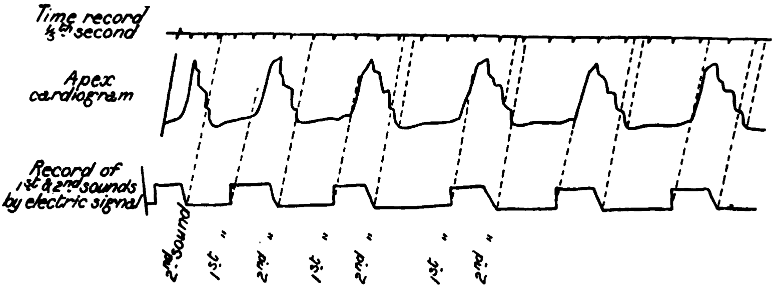


FIG. 158.—COPY OF A TRACING UPON WHICH THE TIME OF OCCURRENCE OF THE FIRST AND SECOND SOUNDS WAS MARKED BY AN ELECTRIC SIGNAL.

It shows that the closure of the semilunar valves takes place about one-tenth of a second before the end of the systolic rise in the tracing. Its time of occurrence is marked by a wave in this tracing. (Not drawn to scale.)

is sufficiently accurate to show that the last wave, before the fall of the lever to normal, marks the point when the semilunar valves close. This point, as measured, is about one-tenth of a second before the final fall of the lever. Thus the cardiogram proves that the extra systole occurs exactly at the end of the expansion period—namely, when the ventricle has filled itself by its own expansion.

This relationship of the extra systole to the normal systole appears to be quite constant in the tracings, and so far as simple inspection can tell there seems to be no variation in its position. The same is true of careful measurements.

Measured Position of the Extra Systole.—On measuring the exact position of the extra systole in relation to the preceding ventricular contraction, it is found that its relationship to that event is constant, thus confirming the cardiographic observation as to its occurrence immediately after the cessation of the cardiac activity due to the preceding beat. Its situation was measured on the cardiogram in eighty instances, and the average time elapsing between the start of the extra systole and that of the preceding beat was 0·446 of a second—a usual length of time for this part of the cardiac cycle. The range of variation in any one series of observations was very small. For instance, thirty extra systoles gave an average of 0·45 second; but out of these in only three instances was the time of occurrence 0·03 second less than the average, and in only four beats was it as much as 0·03 second more than the average.

When the mode of action of the heart was altered by drugs some variation was noticeable.

Thus when taking strophanthus, the extra systole occurred on the average 0·386 of a second after the commencement of the preceding systole.

When under the influence of digitalis, the average was 0·47 of a second. When taking aconite, there was a greater variation, and occasionally the depressing effect of the aconite showed itself by a lengthening of the interval between the commencement of the normal systole and of the extra systole, in addition to the lessening of the frequence of the extra systoles. Thus seven extra systoles out of fourteen in one tracing gave an interval of 0·5 second and two of them of only 0·42 second. The average of the fourteen extra systoles being 0·476 second.

Nevertheless, these variations do not conflict with the statement already made—namely, that the extra systole always occurs at the point where the cardiac activity ceases.

This point marks the end of the expansion phase, and on the cardiogram it is the lowest point reached of the tracing after the systolic rise.

It may, therefore, be said definitely that in this case the extra systole occurred exactly at the end of the expansion phase of the cardiac cycle. In other words, the adventitious contraction does occur just when the ventricle has finished drawing blood into itself by its own active expansion.

PROOF THAT THE VENTRICLES DO FILL THEMSELVES BY THEIR OWN EXPANSION.—This case affords evidence that the major part of the filling of the venticles *does*, as already affirmed, occur during the expansion phase of the cardiac cycle, as was advocated twenty years ago by Dr. Mayo Collier. The fact that an effective contraction can take place one-tenth of a second after the ventricle has emptied itself must be taken as evidence, that, during that one-tenth of a second, the ventricle has been refilled. That the contraction is effective is proved by its power of producing so distinct a wave in the veins of the neck (*vide* fig. 157). That the filling is done by ventricular aspiration is proved by the absence of any auricular contraction whereby the ventricle could be filled, as is shown by the tracings (*vide* fig. 159).

The EXTRA SYSTOLE is due to over-distension of the ventricles.

The occurrence of the extra systole at the precise point when the ventricle has filled itself is strongly suggestive of the probability that a stretching of its muscular walls by over-distension is the cause of the extra systole. If this overfilling did occur and the ventricle could be stimulated to contract by the overstretching of its wall, the contraction would take place precisely at the time when the extra systole does occur in this case. If this were so, there would be three factors at work:—

1. Undue irritability of the heart.
2. Excessive aspiratory force.

3. Abnormal supply of blood, from auricular dilatation or other cause.

The effect of variation in these factors can be studied. Variation in the first factor will not give evidence either for or against this theory. Variation in the second factor will give, however, important evidence if it can be shown that increase of the aspiratory force of the ventricle increases the cardiac irregularity and that diminution of that force diminishes that irregularity.

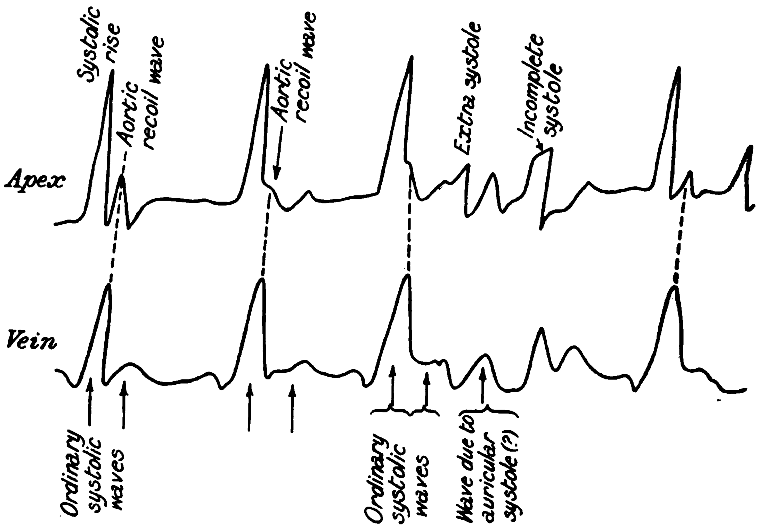


Fig. 159.

This tracing (which is a copy of a portion of that shown in Plate XI, No. 7) demonstrates the fact that there is no sign of any auricular wave preceding the extra systole, and that therefore the fullness of the ventricle with blood (which the occurrence of the extra systole shows) must be due to the ventricle having filled itself by its own expansion. See also fig. 157 for absence of auricular waves in the vein prior to the extra-ventricular systole.

Now, digitalis has the power of increasing the aspiratory force of the cardiac muscle, and aconite, by making the muscular contraction more sluggish, will lessen both the elastic and the muscular recoil of the heart wall after its contraction. Digitalis, therefore, ought to increase and aconite ought to lessen the irregularity of the heart if the theory here advanced be correct. This was found to be the case. Tincture of digitalis in twenty-minim doses every four hours was given, with the result that the irregularity was greatly increased. Unfortunately, some of the tracings taken while the patient was on digitalis have been lost, and the dates were omitted from others. Tracing No. 8, Plate XI, does, however, I believe, show the effect of digitalis and the marked increase of irregularity caused by it. Tracing No. 7, Plate XI, shows a similar type of irregularity, and it was certainly taken during the adminstration of digitalis.

In contrast with this, tincture of aconite in two-minim doses every two hours was given, with the result that the irregularity was very greatly lessened. The action of the aconite in making the beat more sluggish may be seen in the lengthening of the systole from an average of 0·45 second before the drug was given to an average of 0·47 second while under its influence, and for some beats to as much as 0·5 of a second. Under aconite, the frequency of the extra systoles dropped from one in two beats to about one in five beats (e.g., forty-seven in ninety-seven beats and seven in thirty-six beats). Later on tinct. digitalis (♏xx) every six hours was tried again, but again it produced a sort of double extra systole every fourth beat or less. After this, strophanthus was given—which, in my opinion, has much less power of increasing the ventricular expansion than digitalis has (and is therefore of more value in aortic regurgitation than digitalis is)—with the result that the heart again became more regular and the extra systole dropped in frequency to once in nine beats (eight in seventy-two beats), though occasionally it occurred as often as once in four beats (eleven in fifty). The patient also felt much better on this drug, and gradually improved and was discharged. Variation, therefore, in the second factor strongly confirms the theory that the extra systole is due to mechanical stimulation resulting from over-distension of the ventricle by its own aspirating force.

Confirmatory evidence may also be obtained from variations in the third factor—namely, in the amount of blood supplied to the ventricle.

Now the amount of blood supplied to the right ventricle is apt to vary, in consequence of the respiratory movements of the chest. Inspiration naturally tends to draw blood into the thoracic veins, and, the return of this blood being prevented by the valves at the root of the neck, the commencement of expiration naturally tends to increase the pressure upon the blood thus enclosed, and so favour its entry into the right ventricle. This will mainly affect the first beat which occurs after the end of expiration.

This is of course the secret of the well-known relief given by 'the long breath' in anæmic dilatation of the right ventricle.

Moreover, careful measurement of the loudness of the right ventricular sounds demonstrates the same fact; for the pulmonary second sound and the right ventricular murmurs are often measurably louder in the case of the first cardiac beat that occurs during expiration than in the rest of the cycle. These facts show that the first beat to occur after the end of inspiration is apt to be more effective than others, owing to the ventricle being then at its fullest. Now in this patient it was sometimes noticed that the extra systole only occurred at the end of inspiration—namely, as has been shown, when the right side of the heart was extra full of blood and there was the likelihood of an excess of blood being aspirated into the ventricle (*vide* Tracing No. 3, Plate IX). Therefore, variation of the third factor also supports this theory.

ACTION OF THE AURICLES AND GREAT VEINS

This case also throws light upon the activities of the auricle and great veins. My study of venous tracings in this and other cases has convinced me that—in abnormally working hearts, at all events, if not in all human hearts—the auricle does more work than it appears to do when investigated in the physiological laboratory. Tracings taken in one instance from over the auricle itself also confirmed this view, for where there is tricuspid regurgitation the auricle appears sometimes to remain contracted throughout the greater part of the ventricular systole (*vide* Plate XII, No. 9).

This case will, I think, show quite clearly to those who make the necessary effort to think out the tracings, that the auricle is in a state of tonic activity throughout the cardiac cycle, and is prepared to respond by increased tonic activity, or by actual contraction, to any sudden increase of pressure tending to distend it.

But, to demonstrate this fact, the blood which regurgitates from the right ventricle, during the extra systole, must be very carefully followed.

Work Done by the Extra Systole

The extra systole, as already pointed out, does not, as a rule, succeed in forcing blood into the aorta, for it causes no wave in the carotid artery. Therefore, presumably, its energy is expended in forcing blood back into the auricles and veins.

Before going further, it were well to point out that, in obtaining cardiographic evidence as to what is taking place in the heart and veins, it is sometimes necessary to turn to the right side of the heart and sometimes to the left side, upon the hypothesis—which must be accepted—that the two sides of the heart act in unison. All the evidence here points to the fact that they do so act.

Wave in Veins Due to the Extra Systole.—In the jugular bulb, there is a well-marked wave, which I have spoken of as being due to tricuspid regurgitation, the result of the extra systole. Careful measurements prove that this is its true explanation, although, at first sight, it might be easily mistaken for a wave due to an auricular systole: so much so, that an eminent authority on venous tracings, to whom I showed this one, expressed his belief, after superficial examination, that it was probably an auricular wave. Therefore, the proof of its ventricular origin must be gone into somewhat in detail, although it involves a close study of the tracings.

Recoil Waves in the Apex Cardiogram.—If the extra systole be examined it will be seen that the rise it causes in the cardiogram is not a single but a double one. There is in every case a distinct shoulder some distance down the descent of the curve, showing that the outflow of blood is more or less suddenly checked. This arrest causes a wave in the cardiogram which may be well described as a recoil wave (*vide* figs. 160, 161, &c.)

—namely, a wave due to the recoil of the out-flowing blood from some hindrance which it experiences.

Recoil waves of this nature are of usual occurrence on the ordinary systolic cardiogram, if taken a little to one side of the apex beat so that the diminution in size of the ventricle, which results from the outflow of its contents, can be recorded by the cardiograph. Such recoil waves show in the present tracing. Their explanation is simple. To take the left ventricle as an illustration. In the normal systole, as soon as the interventricular pressure equals that in the aorta the blood begins to rush out, and, taking the direction of least resistance, dilates the first part of the aorta. But the momentum of the first rush of blood so over-distends the vessel that its elastic recoil comes into play, and the sudden rise in pressure, so caused, hinders slightly the outflow of blood, because the ventricle has to increase its force so as to overcome this increased resistance. This slight check to the outflow often shows in the cardiogram as a distinct wave. There may be two or more of such waves to be seen (*vide* fig. 160, point 3; fig. 162, point D).

But to return to the extra systole and its recoil wave.

The well-marked recoil wave here shown cannot be due to the elastic recoil of the aorta, for it has been demonstrated that the extra systole is an imperfect contraction and does not send blood into the aorta.

Moreover, it is not due to a recoil from an ineffectual attempt to open the semilunar valves, because it occurs after the ventricle has got rid of a considerable part of its contents, for it occurs nearly half-way down the descent of the curve.

It must, therefore, be due to some hindrance to blood which is regurgitating through the auriculo-ventricular valves into the auricle.

We must now turn to the right side of the heart, because here alone can we study graphically what happens to the regurgitating blood.

But before entering upon the discussion of the various waves in the venous tracing and their significance, it will be necessary to point out that the argument must be based upon exact correlation of the waves in the vein and in the apex tracing by means of accurate measurements.

No Delay in Transmission of impulses between the ventricle and the veins of the neck.—The correlation of venous and apex tracings by measurements is only reliable in so far as it is possible to allow for the amount of delay that takes place in the transmission of impulses from the ventricle to the neck. It might be supposed that there would be delay and that, for instance, the wave due to the auricular contraction would reach the jugular vein a measurable time after it showed over the ventricle. Measurement, on the other hand, shows that there is no recognisable delay at all and that the auricular wave reaches the two spots practically simultaneously (figs. 161, 162). It must be remembered that tracings taken over what is called the jugular bulb, record the movements of the blood which is enclosed in the great veins between the valves at the root of the neck and the heart. Therefore, we can say that the tone of the muscular walls of these veins is so high—in this case, at all events—that the blood within the great veins behaves, so far as the transmission of impulses is concerned, as if it were in a tube with rigid walls. Therefore, in dealing with the transmission of impulses from the heart to the jugular bulb, no time allowance need be made, and the transmission may be considered instantaneous.

But to return to our argument, regarding the nature of the 'recoil wave' on the wave due to the extra systole. There is no evidence of any resistance in the veins which could cause this wave. At the time of its occurrence the veins are almost at their emptiest (*vide* fig. 157; figs. 160 and 161, point 8), and there is no sign of any increased pressure in them adequate to cause such a hindrance to the tricuspid regurgitation as is evidenced by this recoil wave. As a matter of fact this recoil wave exactly corresponds to the lowest point of the venous tracing just before the commencement of the big rise, of which we are about to speak. We are therefore driven to the conclusion that the recoil which causes this wave must come back into the ventricle from the auricle.

Tonic Contraction of the walls of the Auricle during diastole.—This brings us face to face with an important fact—for such it must be called—namely, that the auricular walls are in a state of tonic contraction during the diastole. Now at the point of time of which we are speaking, the auricle has just been completely emptied into the ventricle, and therefore ought to be relaxed and empty and able to receive the greater part of the contents of the ventricle, without offering any resistance to their reception. The fact that it is able to return a well-marked recoil wave into the ventricle before the latter has half-emptied itself proves that the auricular walls must have contracted down as the blood passed out of it, and must remain in a state of tonic contraction and resent the entrance of the blood regurgitating from the ventricle. Now the main wave in the neck of which we are speaking commences immediately after the return into the ventricle of this recoil wave from the auricle, and its maximum rise corresponds to the minimal fall of the apex tracing (*vide* figs. 160 and 161, point 9; and Plate X). It is quite evident from the tracing that this increase in the fullness of the vein coincides with the emptying of the ventricle, and is therefore due to tricuspid regurgitation, the blood being diverted from the auricle into veins by the resistance offered by the muscular tone of the auricular walls. If the large wave in the vein had been due to an auricular contraction there would have been a wave in the ventricular tracing synchronous with that in the vein.

SUDDEN DISTENSION A CAUSE OF AN AURICULAR CONTRACTION

In confirmation of what has just been said there is evidence that such an auricular contraction does take place occasionally.

In some of the heart beats the even descent of the curve, as the ventricle empties itself during the extra systole, is broken by a wave showing a sudden increase of pressure such as would be caused by an auricular contraction (fig. 161, point *a*). In the beats where this occurs the big rise in the vein is broader and more sustained than where it does not occur (Plate X). The appearance of these two synchronous waves may be taken as proof that an auricular contraction occurred at this point (*vide* fig. 161, point *a*; and Plate X).

The cause of this auricular contraction is clear. The hindrance offered to the regurgitation by the auricular walls (to which the recoil wave is due) requires a slight increase in the expulsive force of the ventricle in order to overcome it. This sudden increase in the expulsive force of the ventricle seems in some instances to be too much for the resisting power of the auricular walls and therefore stimulates them to contract. This tracing seems clearly to show that the sudden stretching of the auricular walls which this extra effort of the ventricle occasions, does stimulate the auricle to a true contractile act. In confirmation of this, it will be noticed that, in those beats where this extra auricular systole occurs, the extra ventricular systole is more powerful (if we may judge it by the height of its wave in the cardiogram) than in those beats where it does not occur. Note the relative size of the rise due to the extra systole in the two types of beat in figs. 160 and 161 and Plate X.

But even in the case of these stronger beats the auricular contraction does not *cause* the rise in the venous tracing, but only *augments* the rise which the tricuspid regurgitation is already causing. In those beats where there is certainly no auricular contraction the big wave in the veins is as high, though not so broad, as those where the auricle does contract. The tracings, therefore, show that this rise in the venous pulse is of ventricular and not of auricular origin.

Oscillation of Blood Backwards and Forwards between Veins and Auricle.—In those beats where the blood, regurgitating into the auricle, has not sufficient force to overcome the tonic resistance of the auricular wall, there are other phenomena to be noticed (*vide* fig. 160 and Plate X). In these beats it seems as if the blood were not driven into the veins with sufficient force to overcome the tonic resistance of their muscular walls and therefore this tonic resistance was able to throw the regurgitating blood back once more into the ventricle with sufficient force to make a very distinct rise in the ventricular tracing. Careful comparison of the venous and the ventricular tracings shows how exact is the correspondence between the fall in the venous tracing and the rise in the apex cardiogram, and vice versa. The wave seems to oscillate backwards and forwards, between the ventricle and the veins, before it finally dies down (*vide* fig. 160, points 10, 15, 16).

In those beats where a true auricular contraction occurs, this oscillation of the blood wave is not seen. The reason probably is that the auricular contraction raises the blood pressure in the vein—just as it is commencing to contract—to an extent sufficient to prevent the vein from emptying itself into the ventricle. Note that the wave in the vein which results from the auricular contraction checks the fall which is due to the commencement of the venous contraction. This is well shown in the third beat from the right-hand end of the tracing shown in Plate X. It will be noticed that the larger beats, where the auricular contraction occurs, often alternate with smaller ones where there is none and where two other waves follow the extra systole in the apex tracing and one in the venous tracing.

CONTRACTION OF THE VEINS.—In the smaller beats, already spoken of, the big wave in the veins has another characteristic besides its relative narrowness. If it be compared with the alternate broader ones it will be seen that the emptying of the vein is more sudden and that the fall is often continued below the base level. Both these features imply a degree of suddenness which points to the occurrence of a true contraction of the vein. It is not unusual to observe, in venous tracings, phenomena such as these, which seem to point clearly to the occurrence of true contraction of the veins. The occurrence of this contraction in the veins cannot be recorded graphically except under certain conditions—namely, the distension of the vein, while being sudden enough to produce a well-marked contraction, must not be so great as to overpower the muscles of the veins, nor so prolonged that the stimulus to contract has time to pass off and thus lose its effect, before the pressure is sufficiently lowered to admit of the contraction emptying the vein. In the beats where the extra auricular systole occurred these two conditions were not fulfilled and no evidence of any contraction of the veins is visible.

WAVE IN VENTRICULAR TRACING DUE TO A RETURN WAVE

FROM THE VEINS.—Where, however, this effective contraction of the veins does occur, the blood is returned towards the ventricle with sufficient force to cause the second rise in the apex cardiogram of which we have been speaking.

On looking at this second rise (*vide* fig. 160, points 10, 11, and 12), it will be noticed that the upstroke is rather a gradual one, and sometimes there are two more or less distinct waves—one on the upstroke, and one constituting the apex of the rise. The first of these two waves is synchronous with the emptying of the vein, and is due to the impact upon the ventricle wall of the blood sent into it by the venous contraction. The second is synchronous with a small wave in the venous pulse. This wave must be of auricular origin and produced by the tonic response of its walls to the blood sent into it from the veins. When the veins contract, the blood is thrown into *both* auricle and ventricle, but the tonic response of the auricular wall sends it out again in both directions, and so causes both the small wave in the vein and the highest point of the rise due to the filling of the ventricle (*vide* fig. 160, point 12).

SENSITIVENESS OF VEINS AND AURICLE TO SUDDEN CHANGES OF PRESSURE.—There are one or two other facts which testify to the sensitiveness of the auricle and the great veins to sudden variations of pressure within them, and to the speedy passage of waves to (and in) them, owing to the tonic contraction of their walls. For instance, at the time of occurrence of the maximal fullness of the vein, during the tricuspid regurgitation, there is a small oscillation over the ventricle synchronous with the apex of the venous rise, showing that the increase of tension due to the venous distension shows itself in the ventricle (*vide* fig. 160, point 9; and Plate X).

Again, there is sometimes a small oscillation in the vein synchronous with the commencement of the extra systole, as if the rush of blood from the ventricle into the auricle caused a slight disturbance in the intravenous pressure; but, as it found fairly free entry into the auricle, a more marked wave was not caused in the vein.

I think I have now given abundant evidence to prove the thesis I set out to prove—namely, 'that throughout the diastole the blood in the auricles and great veins is maintained—in this case, at all events—at a definite degree of tension by the muscular walls of both auricle and veins, and that their walls are so sensitive as to respond at once by increased tonic activity or by definite contraction to any sudden tendency to alteration of the blood pressure within them.'

This tracing also seems to show that the veins are stimulated to contract by any sudden lessening of the blood pressure within them. The deepness and suddenness of the emptying, which takes place after the normal auricular systole (during what is, presumably, its time of elastic recoil or active expansion), seems to show that a true venous contraction occurs here.

ADDITIONAL EVIDENCE.—When this patient was taking aconite, and the extra systole was prevented, in most of the beats, by the lessening of the expansile force of the ventricle, there was, nevertheless, evidence that the ventricle was still somewhat over-distended by its own expansile force.

In fig. 162, point *e*, it will be seen that the slight depression in the venous tracing which corresponds to the aspiration period of the ventricle is immediately followed by a slight filling up of the vein. This shows clearly that the blood not only rushes into the right ventricle during its expansion, but also that it more than fills it, and that some passes out again into the veins—as the result, presumably, of the elastic recoil of the over-distended ventricle. In this tracing, however, the over-distension is not sufficient to stimulate the ventricle to contract and cause an extra systole. In this tracing, when the extra systole does occur it causes only a small wave in the veins of the neck, and some of the blood therefore must either simply return into the auricle, or else, in this case, the ventricle must succeed in sending some blood into the pulmonary artery and aorta. Of these two hypotheses, I think the latter the more probable, although, unfortunately, I have not a carotid tracing whereby to verify it. This supposition that the extra systole does pass some blood through the semilunar valves is confirmed by the character of the succeeding ordinary systole, for its commencement is delayed and it also does less work than usual, so far as can be judged from the breadth of the systolic rise in the cardiogram.

Careful measurements of the amount of delay of the start of the systole as compared with the delay in the time of occurrence of the second aortic recoil waves, brings out an interesting fact—namely, although the start is delayed to the extent of nearly one-eighth of the normal interval (i.e. more than 11½ per cent.), the time of occurrence of the second aortic recoil wave is only delayed less than one-sixteenth of the normal interval (i.e. 6 per cent.) (*vide* fig. 162). This observation shows that although the commencement of this systole is much delayed, nevertheless it fills the aorta very quickly. This must, I think, be taken as evidence that the extra systole had already thrown some blood into the aorta and partly filled it. In this way the extra systole, and the normal systole following it, together make up one normal beat. Moreover, there is no lasting interference with the cardiac rhythm, for the next beat after the extra systole occurs at the time it would have done had there been no extra systole. (See also the explanatory notes to fig. 162.)

PROLONGED AURICULAR CONTRACTION IN TRICUSPID REGURGITATION.—This theory of the tenseness and sensitiveness of the auricles and veins is also supported by the only tracings that I have been able to obtain from the right auricle itself. These showed that the auricle was drawn away from the chest wall during its own systole and remained in the same state until nearly the end of the ventricular systole. But the interesting point was that the systolic wave which showed in the ventricular cardiogram was reflected in miniature in the contracted auricle, making the portion of the auricular tracing which corresponded to the ventricular systole resemble the ventricular one to some extent. I could not understand this at the time (namely, in 1892); but now it is plain, for I know that the auricle is sensitive to every change in pressure that takes place in the ventricle (*vide* Plate XII).

TONIC ACTIVITY OF THE VENTRICULAR WALLS DURING THE 'RELAXATION' PHASE (?)

These tracings also seem to demonstrate the fact that the wall of the right ventricle is not in a state of passivity during its relaxation phase, but is in a state of tonic activity, and sensitive to mechanical stimulation by any sufficiently sudden increase of pressure within it. The oscillation of blood between the veins and the ventricle which has just been spoken of, *might* be explicable on the supposition that the blood is returned from the ventricle back into veins by its *elastic recoil* after an over-distension due to the venous contraction.

As the venous contraction *is* sufficiently powerful to distend the ventricle (see point No. 11 in the ventricular tracing, fig. 160), this is a possible supposition. Nevertheless, the suddenness of the fall, due to the emptying of the ventricle, suggests that the more probable explanation is that the ventricle possesses tonic activity. The possession of tonic activity having been proved in the case of the auricle renders probable its possession of the ventricle also. Further study, however, is necessary before this point can be considered to be proved.

This possession of tonic activity by the left ventricle, during its relaxation phase, is also suggested by the suddenness of its emptying when over-distended by its own aspiration under the conditions described in Essay XII, and illustrated by the tracings shown in the figures on pp. 433, 434, 437, 438. Moreover, the tracings shown on pp. 432 and 433 show waves following the suction recoil wave, which are almost certainly due to the oscillation of the blood backwards and forwards between the left auricle and left ventricle.

The explanation of the extra systole, which this case gives, throws much light upon the occurrence of this type of irregularity in valvular disease. This type of extra systole is a very common one, and it is highly probable that when it occurs it shows that the ventricle is being over-distended by its own expansile force. Certainly, over-distension is likely to occur in an embarrassed heart with a dilated auricle, such as is frequent in valvular disease.

Further consideration of this and similar cases will, I have no doubt, bring out the fact that the inception of cardiac contraction is not such a purely spontaneous function of the various nodes as some writers seem to think, but that the occurrence of cardiac contraction may also often be of the nature of a reflex act, the afferent impulses being caused by the distension and stretching of the cardiac walls. It is probable that the occurrence of reflex cardiac contraction will be proved before long by means of the electric cardiograph.

EXPLANATIONS OF THE TRACINGS

FIG. 160.

Two portions of the tracing given in Plate X are here shown. Each portion includes a single normal ventricular contraction with an extra systole and the wave due to the return of blood to the ventricle again from the veins.

In each portion, the ventricular and the venous tracing are correlated by means of thin vertical lines connecting the corresponding points of the two tracings. These lines are copied from the original tracing in which the points in the ventricular and the venous tracing were very accurately ascertained. The tracing given in Plate X will enable anyone, who is desirous of doing so, to verify this statement by actual measurements.

In the first portion the main events recorded are indicated in words.

In the second portion another similar beat is shown, but the various distinct events are each numbered, in order that they may be described in detail, as follows:—

1. The final rise of the auricular wave in the vein is shown to be exactly synchronous with the commencement of the ventricular systole—showing no delay in the transmission of the auricular wave to the jugular bulb, over which the venous tracing was taken.

2. The highest point of the ventricular systolic rise is shown to correspond with the deepest point of the notch which follows the auricular wave in the vein. In this particular beat the suddenness of the drop shows that there was very rapid emptying of the vein. This sudden drop in the tracing suggests that an actual contraction took place in the vein, and supports the theory that the relief of tension which occurred as the blood rushed into the auricle (as it expanded after its systole) was sufficiently sudden to cause a true contraction of the walls of the vein.

3. The first aortic recoil wave—due to the check which the blood flowing out of the ventricle receives when the first part of the aorta is fully distended. This point coincides with the highest point of the systolic rise in the vein. This fact seems to prove that the systolic rise in the vein is—in this patient, at all events—due to the compression of the superior vena cava by the over-distension of the first part of the aorta. (This aortic recoil wave does not show very clearly in this beat, but it does show in the first beat given in this figure.)

4. The second aortic recoil wave. This wave appears to be due to the hindrance to the outflow of blood from the ventricle, which results when all the main arteries are distended and the elasticity of their walls begins to come into play, and thus necessitates a final expulsive effort on the part of the ventricle.

This second recoil wave is more often evident upon the ventricular tracing than is the case with the first.

In the venous tracing this recoil wave often coincides with the point where the fall due to the emptying of the vein after its systolic fullness comes to an end.

According to the theory just given as to the cause of the systolic rise in the venous tracing, this is to be expected; for the fall is due to the relief of pressure on the superior vena cava which accompanies the emptying of the first part of the aorta as its elastic recoil comes into play, and the return of blood towards the heart from the distant arteries will again increase the amount

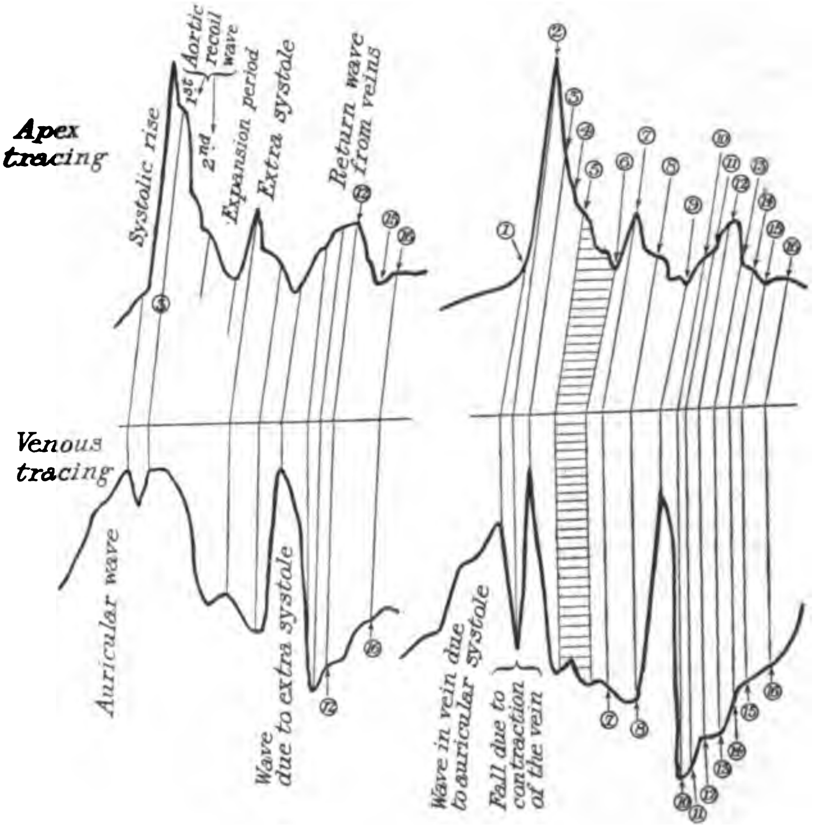


FIG. 160.

of distension of this part of the aorta and so tend once more to compress the superior vena cava somewhat.

5. This wave in the ventricular tracing marks the time of occurrence of the second sound of the heart and the commencement of the expansion phase.

In the vein it sometimes coincides with a somewhat sudden drop due to an emptying of the vein at the time when the blood is being aspirated from the auricle into the ventricle. This is shown in fig. 161.

More often, however, in this particular case, the fullness of the jugular bulb does not appear to be materially lessened at the time of the ventricular aspiration (*vide* fig. 162).

6. Marks the lowest point of the ventricular tracing and the termination of the expansion phase.

In the normal beat it marks the point where relaxation commences, but here it marks the commencement of an abnormal contraction—namely, the extra systole.

In many of the tracings this point falls part-way up a small rounded rise in the venous tracing. It will be remembered that we are dealing here with a case in which the ventricle is presumably aspirating more blood towards itself than it can well hold. A certain amount of this blood may therefore be expected to escape from the ventricle again when the elasticity of the ventricle wall comes into play. Such a return of blood from the ventricle into the veins probably causes the small rounded rise in the venous tracing, at the commencement of the relaxation phase, of which we are now speaking.

7. The highest point of the rise in the ventricular tracing which is due to the extra systole. In the vein, this corresponds to the highest point of the rounded rise just referred to.

8. The recoil wave which causes a break in the fall of the ventricular tracing after the rise due to the extra systole. This must be due to some check to the outflow of blood (as pointed out, p. 503). It must be due to the tonic contraction of the walls of the auricle which is caused by the inrush of blood from the ventricle. This recoil wave is shown to correspond with the lowest point of the venous tracing just before the big rise.

9. This is the lowest point of the ventricular tracing, and corresponds to the highest point of the big rise in the venous tracing which is due to the distension of the veins by the blood which enters them as the result of the extra systole.

In this tracing, as pointed out at p. 505, it appears that the tonic response of the walls of the auricle drives the blood which has entered it out again into the veins.

10. Marks the foot of the fall due to the emptying of the vein after its distension.

Here the fall is sudden enough to suggest a true contraction of the walls of the vein, their sudden over-distension presumably stimulating the muscles of their walls to a true contraction. The emptying of the vein coincides with a re-filling of the ventricle (*vide* point 10 on the ventricular tracing).

11. At the point 11, a further rise takes place in the ventricular tracing, and this also coincides with a rise of the venous tracing. This sudden slight filling of both vein and ventricle must be due to auricular activity, and the explanation is that the inrush of blood into the auricle caused a tonic muscular response, and this drove the blood out again into both vein and ventricle.

12. The maximal point of this rise in both vein and ventricle.

13. The ventricular rise due to this return of the blood from the veins now suddenly subsides and the blood is once more driven back into the veins, but with lessened force.

After this there is a slight return once more to the ventricle, and sometimes another wave shows in the vein before the oscillation of blood between the ventricle and the jugular bulb finally subsides.

15. Marks the lowest point in the ventricular tracing and corresponds to a slight crest in the vein, and, again—

16. Marks the reverse, a slight crest in the ventricle tracing, corresponding to a slight depression in the venous one.

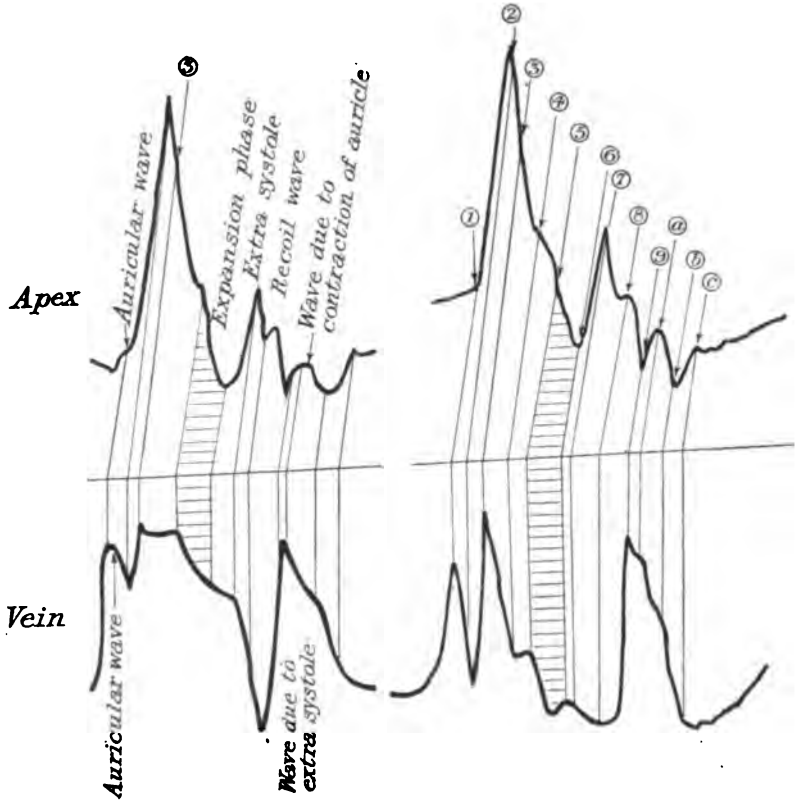


FIG. 161.

This is another portion of the tracing given in Plate X.

The explanation given of fig. 160 also applies to this figure up to point 9.

At 9 in this tracing the fall of the tracing as the ventricle empties itself at the end of the extra systole is checked by the occurrence of a fairly sharp wave with a rounded top. (Wave (*a*).)

(*a*) This is due to a distension of the ventricle by a true contraction of the auricle, the result of its sudden distension by the regurgitation which the extra systole causes.

Note that in the beats where this auricular contraction occurs the extra systole (as judged by the tracing) is more powerful than in beats (such as those shown in fig. 160) where there is no auricular contraction.

In the jugular bulb tracing there is a broadening of the wave when this auricular contraction occurs (see Plate X).

(*b*) The lowest point of the ventricular tracing corresponds to an increase in the distension of the vein (as judged by a broadening of the big rise in the venous tracing) as the blood which the auricle sent into the ventricle is again sent out of it into the vein.

(*c*) This is a small pointed wave in the ventricular tracing which corresponds to the final emptying of the vein after the big rise due to its distension by the extra systole. The fact that no wave in the vein accompanies it negatives the possibility that it is due to a return of blood back from the auricle, and the fact that no further oscillations take place after it points to its being an expansion of the ventricle following the contractile effort which caused the sudden drop after wave (*a*).

Fig. 162

This is a portion of Tracing No. 6, Plate XI, which has already been referred to at p. 508, and which shows tracings taken while the patient was under the influence of aconite. The upper tracing was taken from the cardiac apex, and the lower from the jugular bulb. Corresponding points upon the tracings are connected by thin vertical lines. The length of time intervening between various points of the tracing in each successive beat is shown (in decimals of a second) by the figures upon the thin horizontal lines drawn between those points.

The following facts are to be noted :—

1. There is no well-marked wave in the vein, directly due to the ventricular extra systole, such as is shown in figs. 157, 160, 161. If present it ought to occur at point A.

2. The narrowness of the ventricular rise in the apex tracing, due to the systole which follows the extra systole—i.e. point B. This may be due to exhausted excitability, or to some blood having been passed into the aorta by the extra systole, thus leaving less than the normal amount of work for the ordinary ventricular systole to do.

3. The rise in the venous tracing which corresponds to this same ventricular systole takes place later than usual, and it corresponds to the second aortic recoil wave instead of the first, as is the case normally. Compare point C with point D.

According to the theory here given as to the cause of the systolic rise in the vein, this lateness of the rise, in this instance, points to the first part of the aorta not being fully distended in the early portion of the ventricular systole, but to its full distension at the time of the second aortic recoil wave. This might be expected to occur when (as is probably the case in this particular beat) the ventricle contains less blood than normal, owing to some blood having been already passed on into the aorta by the extra systole.

4. The wave in the vein which occurs at the end of the ventricular expansion period (point E on the tracing), and is due to a reflux of blood from the ventricle (consequent upon its overfilling itself during its expansion period) is smaller when the extra systole occurs than in the normal beats. The explanation of this is to be found in the fact that the extra systole is, in this instance, an effective contraction, closing the auriculo-ventricular valves at the termination of the expansion period and passing the ventricular contents —in part, at all events—through the semilunar valves (i.e. the ventricle in this beat has less blood to deal with, it is less overfilled by its own aspiration, and there is, consequently, less reflux into the veins afterwards).

5. The vein shows a greater degree of emptying than normal during the expansion period of the beat which follows the extra systole. This is evident on comparing the point F with the tracing just before the point E in the preceding beats.

The explanation of this is to be found in the fact that, the ventricle being

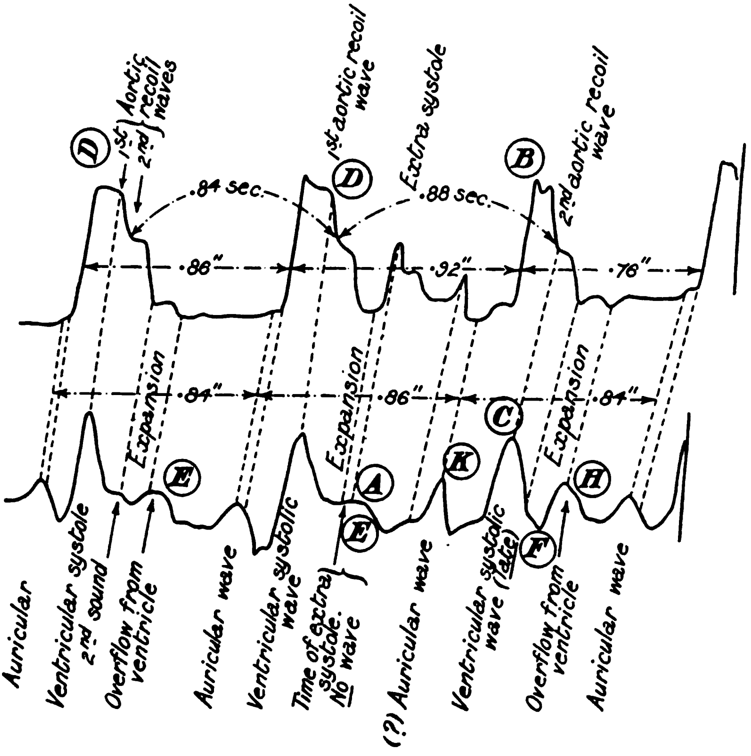


FIG. 162.

imperfectly filled, its beat will end more suddenly than normal. This will increase the force of its expansion and thus draw more blood than usual from the auriculo-venous reservoir.

6. The wave in the vein, due to the return of blood from the ventricle, at the end of the expansion period is higher than normal in the beat following the extra systole. Compare the height of the wave at the point H with the two preceding beats at the point marked E.

This is due to the increased aspiratory force spoken of in paragraph 5. The abnormal amount of blood drawn towards the ventricle will cause an abnormally high wave when, on failing to gain entrance into the ventricle, it returns back into the vein.

The wave K in this tracing is of somewhat doubtful origin.

In the first place it occurs at the time an ordinary auricular wave in the vein is to be expected, as shown by the measured distances on the tracing. Moreover, a precisely similar wave in the vein, which occurs in the tracing shown in Plate IX, No. 4, *is* synchronous with the 'auricular wave' in the apex tracing, and must, therefore, be considered for certain—in part, at all events—an auricular wave.

In the present instance, on the other hand, this wave K occurs a measurable time before the next ventricular systole, and, also, there *is* in the ventricular cardiogram evidence of an auricular wave bearing the normal relationship to the systolic rise B.

If must, therefore, be admitted that this rise K precedes the auricular systole, and, also, that in this particular beat the auricular systole does not cause a wave in the veins of the neck.

There are two factors which may assist in explaining this somewhat unusual phenomenon of an auricular systole being unaccompanied by a wave in the jugular bulb.

Firstly, the vein is just contracting down in the act of expelling the blood which caused the rise K, and its muscular tone must, therefore, be high at this particular moment; and, secondly, there is less blood than usual for the auricle to deal with and the ventricle is less full than normal, and, therefore, the auricular contraction will have to develop less force than normal. These two factors may be sufficient to explain the absence of a distension of the jugular bulb at the time of the auricular systole.

With regard to the work done by the extra systole in this type of tracing, evidence is given by the latter part of Tracing No. 4, Plate IX, which confirms the statement that the extra systole does throw some blood back through the tricuspid valve (into the auricle or the veins or both) as well as some blood forwards into the pulmonary artery, and that what is apparently a normal auricular wave in the vein must—in part, at all events—be due to tricuspid regurgitation.

In the earlier part of Tracing No. 4, Plate IX, the 'auricular' wave, which is nearly synchronous with the end of the extra systole, is but little larger than is usual in this type of tracing. This is so in the portion of No. 6, which is copied to make fig. 162. In the second extra systole of No. 4, however, the wave in the vein is decidedly high.

At the end of No. 4 the type of beat changes and the heart adopts a new rhythm, in which an extra systole follows each beat.

Now in the last beat, prior to the adoption of the new rhythm, an extra systole occurs which is similar in all respects to those seen in the earlier part of the tracing and the interesting point is that this extra systole is accompanied by the wave K in the vein (of which we have just been speaking), although there is no succeeding ventricular systole and no evidence of any auricular contraction. Therefore, this particular beat suggests that tricuspid regurgitation does play an important part in the production of this wave K, which would ordinarily be described as being a normal auricular wave in the vein.

The wave in the ventricular tracing which follows the extra systole is probably due to ventricular expansion, and is discussed at p. 520.

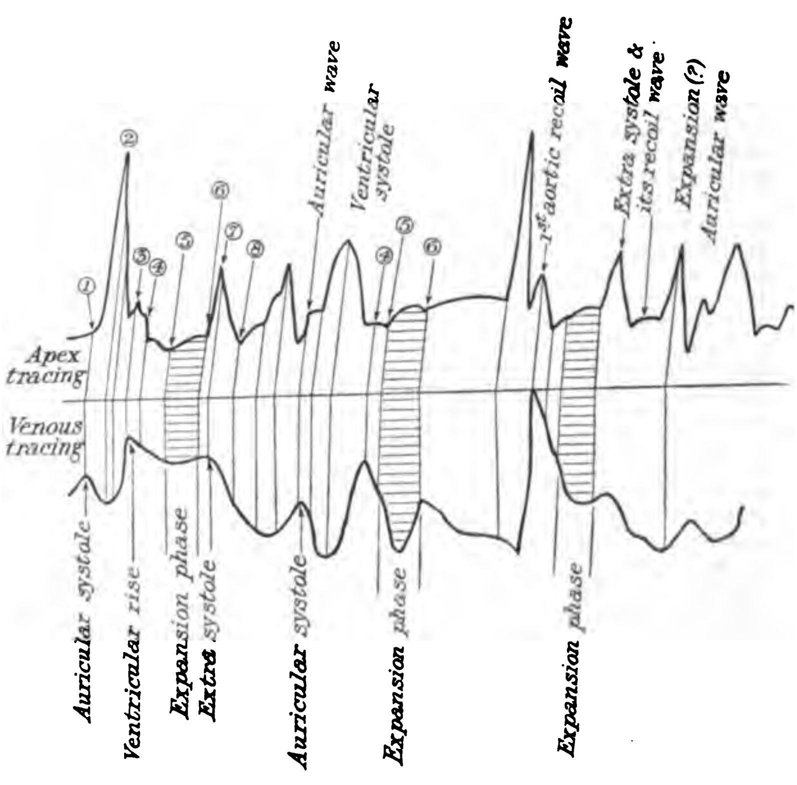


FIG. 163.

This is a portion of Tracing No. 8, Plate XI.

This tracing, showing the result of digitalis, is a good illustration of what has just been said as to the danger of accepting an interpretation of a tracing which does not suffice to explain every individual wave of the tracing.

This tracing at first seems to illustrate the passage of a wave to and fro between the veins and the right ventricle, just as is the case with fig. 160; but this interpretation does not apply to the first rise in the ventricular tracing which follows the extra systole, and more careful study shows that this is quite a different type of tracing from that shown in fig. 160. Comparison of the venous tracing with that shown in fig. 162 and Tracing No. 4, Plate IX, shows clearly that the last wave of the three small ones which follow the normal systole is really an imperfect true ventricular systole and that the venous wave which precedes it is the normal auricular wave occurring in its proper position and proper relation to the ventricular systole. The extra systole—as might be expected from the effect of digitalis—is much more powerful, and the emptying of the ventricle so sudden, that its recoil wave only shows on the base level of the tracing, and sometimes it even forms part of the rise of the wave which follows the extra systole. Both these waves have no corresponding wave in the veins, and therefore it must be concluded that in this instance the extra systole does not force blood into the veins as is the case in the tracing shown in figs. 160, 161.

The venous tracing is normal except that the systolic rise, due to the filling of the first part of the aorta, is later than normal, and occurs at the time of the second and final aortic recoil wave instead of the first.

The pointed wave with a sudden drop, which follows the extra systole, is not very easy of explanation by means of this tracing alone. The fact that it coincides with the lowest point of the venous tracing, and that there is no oscillation in the vein corresponding either to it or to the extra systole, suggests that no blood is forced into either the auricle or the vein by the ventricular activity which causes these two waves.

In this instance, therefore, the extra systole must be forcible enough to open the semilunar valves and empty the ventricle in the normal way. Unfortunately, in the absence of a carotid tracing this point cannot be actually proved.

There can, however, be little doubt that in this case the extra systole is not only forcible enough to open the semilunar valves, but also to cause a true muscular expansion of the ventricle, and this expansion movement is, in all probability, the cause of the rise which follows that due to the extra systole.

Note that this tracing is taken so far internal to the main axis of the heart that the change in volume which accompanies the emptying of the ventricles causes a marked drop in the cardiogram. (Compare this tracing with those of the type given in Plate IX, No. 2, which are taken over the main axis of the heart and where the systolic rise, due to the hardening of the cardiac muscle, persists so long as the muscle remains active.)

There is another feature of this tracing which seems to support what has just been said as to most of the blood being thrown forward through the semilunar valves by the extra systole instead of back into the veins—namely, the fact that the wave in the vein caused by the auricular contraction is always very small and is sometimes hardly visible. This points to the fact that there is no abnormal excess of blood to be dealt with by the right auricle such as would be certain to occur if the extra systole *did* throw the blood backwards through the tricuspid valve instead of forwards. (Compare the size of the auricular wave in the vein in fig. 160, where the extra systole *does* result in tricuspid regurgitation.)

The extreme smallness of the wave due to the auricular systole is also to be expected here, because of the increased aspiratory power which digitalis induces. The ventricle is so well filled during its expansion phase that there is but little work left for the auricular contraction to do. It must, however, be noted, on the other hand, that the wave in the vein which is synchronous with the auricular contraction is decidedly larger in those beats where the extra systole occurs than in the normal beats where it does not. Therefore, whether this particular wave be due to the auricular systole, or to tricuspid regurgitation, or (as is most probable) to both combined, the increase in its height when an extra systole occurs shows that some tricuspid regurgitation must—even in this type of tracing—result from the extra systole.

EXPLANATION OF PLATE XII

This figure shows a tracing taken over the right auricle compared with a ventricular one, from the patient with extreme tricuspid regurgitation whose case is briefly referred to at p. 339. Ventricular cardiograms from the patient are given in Plate VIII, Case V, which show a very marked elevation in the cardiogram during the expansion phase; and in tracings taken near the sternum the highest wave in the tracing occurs just after the occurrence of the second sound.

In order that the somewhat unusual ventricular tracing here shown may be better understood, a somewhat similar but more usual one is given in Tracing No. 10, upon which was recorded the time of occurrence of the first and second sounds by means of an electric signal.

This tracing shows that the main rise is due to the ventricular systole; that the second sound occurs prior to the occurrence of the second rise; and that this elevation is therefore due to the sudden enlargement of the ventricle as it fills itself during its expansion phase.

The irregularity of the heart rhythm made it difficult to time the first sound, but the second was timed with fair accuracy and falls at or just before the highest point of the second main rise.

The two small waves which precede the systolic rise in the tracing must be of auricular origin and presumably represent the contraction of the body of the auricle followed by that of its appendix. (That they are auricular in origin is shown by the fact that their distance from the succeeding systolic rise is constant, whilst that from the preceding one is variable.) On comparing this tracing with the ventricular one shown in Tracing No. 10, there is sufficient resemblance to make its interpretation possible.

The two auricular waves show in both tracings, and the hollow after the main systolic rise occurs in both, as does the second prominence due to ventricular expansion.

In Tracing No. 9, we can therefore say that the first sound would occur towards the foot of the main rise, and that the second sound would occur just at or before the apex of the second rise in the ventricular cardiogram, and the end of the expansion phase would be, as usual, at the foot of the following fall in the tracing.

As regards the interpretation of the auricular part of the tracing it is necessary to point out that this same type of tracing was obtainable all over the pulsating area to the right of the sternum and with both Galabin's cardiograph and a Marey's drum. Also in some tracings the fall which follows the main rise did not, as here, occur before the auricular contraction, but was synchronous with the auricular waves in the apex cardiogram and was broken into two portions—one due to the contraction of the body of the auricle and one due to its appendix. These tracings have unfortunately been mislaid and cannot therefore be reproduced here.

The phenomenon, however, for which this tracing is here reproduced, shows clearly—namely, a small pointed wave in the auricular tracing synchronous with the commencement of the ventricular systole. The auricle begins either to fill up, or to contract again, at the commencement of the ventricular contraction, then it contracts down again, causing a fall in the tracing, and it does not commence to be over-distended by tricuspid regurgitation until more than half the systole is over.

Considering that in this case the loud murmur due to the tricuspid regurgitation, and the distinctly palpable thrill which accompanied it, could be clearly observed at the *very commencement* of the systole it is quite evident that the anterior wall of the auricle (over which the tracings were taken) must have been powerfully contracting throughout more than one half of the ventricular systole in order to attempt—albeit vainly—to check the reflux through the tricuspid orifice.

Note that the main rise in the aricular tracing does not always have the same relationship to the rise in the ventricular tracing which is due to its expansion phase. This is doubtless due to the amount of tricuspid regurgitation varying in the different heart beats here recorded.

SUMMARY OF FACTS (F.) AND THEORIES (T.) DEDUCIBLE FROM THE TRACING SHOWN IN PLATE X

(A) CAUSE OF THE EXTRA SYSTOLE

1. (F.) An extra ventricular systole is interpolated between ordinary cardiac beats without otherwise altering the cardiac rhythm.

2. (F.) There is no sign in the venous tracing of any auricular contraction preceding or accompanying the commencement of the extra systole.

3. (T.) Therefore, the extra systole does not appear to be preceded by an auricular systole.

4. (F.) Whenever the extra systole occurs its time relationship to the beat which precedes it is always the same. It occurs immediately after the cessation of the cardiac activity due to the preceding systole. The fresh rise in the cardiogram immediately follows the termination of the rise due to the preceding ordinary systole.

5. (F.) The extra systole occurs at the end of the expansion phase. This is confirmed by the record of the time of occurrence of the second sound upon the cardiogram.

6. (T.) The occurrence of the extra systole at the exact point when the ventricle has filled itself by its own expansion suggests the probability that this adventitious contraction is due to the sudden stretching of the walls which an *over-filling* of the ventricle would cause.

Over-distension of the ventricle during the expansion phase is evidently the cause of the extra systole in this case.

7. (T.) Two factors are probably at work in producing this over-distension. Firstly, an over-excitable heart giving rise to an excessive aspiratory force, and secondly, dilated auricles supplying an excessive amount of blood at the time the ventricular suction occurs.

8. (T.) If this extra systole be due to the above cause, drugs such as digitalis, which increase the aspiratory power of the ventricles, ought to increase the arhythmia, and drugs such as aconite, which lessen the vigour of the heart's action, ought to lessen the arhythmia.

9. (F.) The arhythmia was markedly increased by digitalis, and markedly lessened by aconite.

(B). FACTS AND THEORIES RELATING TO THE COMPLETENESS OF THE EXTRA SYSTOLE, AND ANSWERING THE QUESTION 'DO THE MUSCULI PAPILLARES AND THE CONSTRICTOR FIBRES OF THE AURICULO-VENTRICULAR ORIFICES PARTAKE IN THE VENTRICULAR CONTRACTION, SO FAR AS THE TRACING SHOWN IN PLATE X IS CONCERNED?'

10. (F.) The extra systole causes as a rule *no* distinct wave in the carotid arteries, and is *always followed* by a very distinct wave in the veins, which often constitutes the highest wave in the venous pulse. Its main effect, therefore, in this type of tracing is to cause tricuspid regurgitation.

11. (T.) Therefore, the mitral and tricuspid valves do not close properly (we must assume that the two sides of the heart act similarly).

12. (T.) It is probable, as the contraction simply results from stretching of the muscular walls of the ventricle (and is not due to the normal spread of stimulus from the auricle), that the stimulus to contract does not involve both the constrictor fibres of the auriculo-ventricular orifices and the musculi papillares in due proportion.

WORK DONE BY THE EXTRA SYSTOLE

13. (F.) In the tracing due to the extra systole there is a recoil wave similar to the one in the ordinary systolic apex tracing (which is due to the recoil of blood into the ventricle from the first part of the aorta after the first sudden distension of the artery).

14. (T.)

(*a*) This recoil wave is not due, in the case of the extra systole, to distension of the aorta, because (F.), as we have shown, the extra systole does not as a rule cause any wave in the arteries.

(*b*) It is not due to the ineffective attempt to open the semilunar valves, because (F.) the tracing shows that the recoil wave occurs after the ventricle has already considerably diminished in volume owing to the expulsion of part of its contained blood. (It occurs one third to half-way down the wave, due to the extra systole.)

(*c*) It is not due to a recoil from over-distension of the venous system by the regurgitating blood, because (F.) the shoulder of the apex tracing marking the recoil wave distinctly precedes the filling up of the veins (F.). (The recoil wave in the apex tracing exactly corresponds to the lowest point of the venous tracing which precedes the rise just referred to.)

(*d*) Therefore, the recoil is due to the return of a wave from the auricle back to the ventricle.

(C) FACTS AND THEORIES REGARDING THE MUSCULAR ACTIVITY OF THE AURICLES AND GREAT VEINS DURING THE CARDIAC DIASTOLE, DEMONSTRATING THE FOLLOWING PROPOSITION

Study of this case seems to show conclusively that there is no true relaxation or passivity of the muscular walls of the auricles and great veins, but that throughout the diastole they maintain, by tonic contraction, a definite degree of blood pressure, and are ready to respond at once, by increased tonic activity, or by contraction, to any tendency to undue distension of the auricle or the veins by any sudden inrush of blood.

15. This proposition is supported by the fact brought out in paragraph 14. The sudden recoil of the blood from the auricle suggests tonic activity on its part.

16. (T.) Also the fact that the extra systole causes such a marked wave in the veins shows that either (1) the tonic activity of the auricle is sufficient to resist the inrush of the regurgitating blood from the ventricle and cause its

diversion into the veins, or (2) that the auricle first dilates under the distending force of the regurgitation, and then by a definite act of contraction expels the blood into the veins.

In some cases (1) seems to be the explanation, because (F.) the rise in the vein exactly corresponds to the fall in the apex tracing, and there is no evidence of any increase in the intraventricular pressure such as would be sure to occur were a definite contraction of the auricle to take place.

In other instances (2) seems to be correct, for (F.), a definite wave, does sometimes appear in the apex tracing (closely following the fall of the wave after the extra systole), such as would be caused by an auricular contraction, and this wave coincides with the distension of the vein just as would be the case were that distension due to an auricular systole aiding the regurgitation from the ventricle.

17. (F.) In some beats a second rise occurs in the apex tracing after the extra systole, due to the return of the blood wave back from the auricle and veins into the ventricle once more. A sort of to-and-fro oscillation of the blood between the veins and the ventricle.

18. (F.) In those beats, however, where the presence of the wave discussed in 16 (2) appears to indicate an auricular contraction, this second oscillation does not occur; the increased pressure in the vein (due to the auricular systole) sufficing to overpower the venous contraction and so prevent further oscillation. Moreover, in these same beats the shape of the wave in the veins as a rule suggests a greater amount of distension than in the beats where there is no evidence of an auricular systole. The distension is more prolonged and there is often more or less of a recoil wave or shoulder on the wave in the venous tracing.

19. (F.) In the beats where there is no suggestion of an extra auricular contraction following the extra systole there is evidence (*vide* 16, 1) that the tonic activity of the auricle alone causes the deflection of the blood into the veins. In this case there is sometimes a second marked elevation in the apex tracing. In such a case the vein seems to empty immediately, after the rise referred to, with a suddenness and completeness suggestive of a contraction of its walls, and in such a case

this emptying coincides with a redistension of the ventricle; the wave being reflected back again, as it were, and the maximal fall in the vein corresponds to a point just before the maximal rise of the apex tracing (*see third beat from the end of the tracing*).

(T.) Here again there is further evidence of tonic activity of the auricles, for the rise in the apex tracing gradually attains a maximum. The contraction of the great veins forces the blood into the tonically contracted auricle and the relaxed but nearly full ventricle. The blood forced by the venous contraction into the auricle calls out a slight muscular response, and thus the maximal rise of the wave as shown in the ventricle is only attained just *after* the emptying of the veins.

20. (F.) In addition to these primary waves there are also three small oscillations which also show how the blood between the veins of the neck and the heart apex is practically a continuous column under the same pressure.

(*a*) At the time of the occurrence of the maximal wave due to the extra systole, there is sometimes a slight oscillation, exactly synchronous with it, in the blood in the veins of the neck, then at their emptiest ((T.) owing to ventricular aspiration of the blood from the auricles and veins).

(*b*) In the apex tracing there is often a small wave just after the extra systole, exactly synchronous with the maximal filling of the vein, (T.) as if this distension sent a shock down into the ventricle.

(*c*) At the time of the maximum of the large wave in the apex tracing, which sometimes follows the extra systole, there is often a small wave in the nearly empty vein exactly synchronous with it. (T.) Another recoil wave from distended ventricle to vein.

PHYSIOLOGY OF THE AURICLES AND VEINS AS GIVEN IN ESSAYS IX AND XVII

Sequence of Events in the Auricles and Veins throughout the Cardiac Cycle.—In view of the facts brought out by these and similar observations upon the heart, it is possible to rewrite the sequence of changes which take place in the auricle and veins throughout the cardiac cycle.

Taking for granted that the ventricle *does* fill itself by its own aspiration, the following would be the sequence of events in the auricle.

The end of the ventricular contraction, just prior to the closure of the semilunar valves, would, according to this theory, find the auricle at its fullest; containing very nearly the full charge of blood (say, some 70 c.c. out of the 90 c.c., which represents the normal charge for the ventricle).

Expansion phase of the ventricles.—On the sudden expansion of the ventricles this amount (70 c.c.) is aspirated *into* the ventricle, the $\frac{1}{10}$ second during which the expansion lasts sufficing for this. In the auricle this will cause a sudden relaxation of its walls. This sudden lessening in the tension of its walls will, in all probability, cause a slight muscular contraction, which will aid the entrance of the last portion of the 70 c.c. into the ventricle. The fact of the auricular walls contracting down upon the receding blood, together with the tonic contraction of the auricle and great veins, will keep the blood in the ventricle and prevent its return back into the auricle and the veins. The auriculo-ventricular valves may also aid in the retention in the ventricle of the aspirated blood. This main emptying of the auricle does not appear to be accompanied by any powerful contraction of its walls. Such a contraction would not be desirable, for it would be followed by an expansion phase, which would tend to draw the blood out of the ventricle back into the auricle.

Gradual Filling of the Auricle.—The auricle now gradually fills with blood during the remainder of the ventricular diastole—namely, its relaxation phase—until the onset of the auricular contraction.

Degree of Fullness of the Auricle at the Time of its Systole.—Taking for granted that the flow of blood into the auricles is fairly regular throughout the cardiac cycle, it is easy to estimate the fullness of the auricle at the time of its systole. Taking the whole cycle as lasting one second, in a heart beating sixty per minute, the whole of the ventricular relaxation phase lasts about 0·52 second; but from this must be deducted the $\frac{1}{10}$ second that the auricular systole lasts. This gives 0·42 second as the time during which the auricle is filling, and therefore during this time it will have received 42 per cent. of its full charge—namely, about 38 c.c. out of the 90 c.c. During the filling, its walls have been quietly distending, and its muscles gradually giving way under the distensile force of the entering blood.

Auricular Systole.—Now comes the main auricular contraction. The auricular systole must, according to this line of argument, be purely a ‘nodal’ phenomenon, because the fact that the auricle is only partly full negatives the possibility that mechanical stimulation (by distension) plays any part in it.

The auricular systole takes place in two stages. First the general wall of the auricle contracts so as to raise the blood pressure within it. This initial contraction of the auricle does not always seem to have the effect of forcing blood into the ventricle or affecting its blood pressure sufficiently to make a wave appear in the apex cardiogram. Sometimes, however, a wave does show and then it is possible that, in these cases, some blood is forced into the ventricle by this first part of the auricular systole. In the venous tracing, on the other hand, this initial contraction does, as a rule, show as a distinct wave, and also in tracings taken over the body of the auricle it is very distinct and, in fact, embraces nearly one-half of the drop in the tracing due to the auricular systole.

The function of this preliminary contraction is to prepare for the more powerful contraction of the appendix by fixing the walls of the auricle and raising the pressure within it.

CONTRACTION OF THE APPENDIX

Then follows the second part of the auricular contraction—namely, the contraction of the appendix. The result of this is to force a small quantity of blood (possibly some 20 c.c. or so) into the nearly full ventricle (as described by Dr. Mayo Collier in 1889) in order finally to distend it and put the auriculo-ventricular valves on the stretch just before they begin to bear the strain of the ventricular contraction. The auricular contraction has almost certainly another important function—namely, to precipitate the contraction of the ventricle by means of the mechanical stimulation of its muscular walls which will result from their sudden stretching. This sudden stretching provides 'the spark that fires the powder.' In this connection it is of interest to remember that one of the principal results of auricular fibrillation is *irregularity* in the time of occurrence as well as in the strength of the ventricular contraction. This fairly forcible contractile act naturally makes a wave of considerable size in the veins of the neck, and also causes a slight one in the apex cardiogram at the foot of the systolic rise.

AURICULAR ASPIRATION AND CONTRACTION OF THE VEINS

This contraction of the appendix is, presumably, followed by the usual elastic and muscular recoil, and thereby develops a certain amount of aspiration, as may be seen by the venous pulse. As the auriculo-ventricular valves are closed, this aspiration (in which the body of the auricle may or may not take a part) expends such force, as it develops, upon the blood entering the auricle from the veins and causes a sudden relief of pressure in them. As the veins are at the time distended, in consequence of the reflux caused by the auricular systole, the change is a sudden one, and this sudden transition from full distension to relaxation causes a marked drop in the venous tracing. In the tracings here shown the suddenness and depth of this drop seem to show clearly that the sudden relaxation of the stretched walls of the vein stimulated them to make a definite contractile act. Such a contractile act at this point in the cardiac cycle will distinctly favour the filling of the auricle and prepare it for supplying the blood needed by the ventricle during its expansion phase.

VENTRICULAR SYSTOLE

Then follows the ventricular systole. With regard to its origin this case seems to show that the stimulus which causes the normal ventricular systole is not wholly a 'nodal' one, but that mechanical stimulation by distension probably plays some part.

SYSTOLIC WAVE IN THE VENOUS PULSE

The next phenomenon to be discussed concerns the first part of the aorta and its relation to the superior vena cava. Bearing in mind what has already been said about the degree of tone present in the veins and the fact that any local alteration in pressure will cause an immediate general effect, it will be seen that the sudden pressure which the distension of the first part of the aorta must exert upon the superior vena cava might be expected to cause an impulse in the veins. The tracings seem to prove that the systolic distension of the first part of the aorta *does* cause the systolic rise in the veins. The aortic recoil wave in the ventricular tracing has already been referred to, and its causation by the elastic recoil of the first part of the aorta after its maximal distension has been pointed out.

Now, on comparing the systolic rise in the vein with the time of occurrence of this recoil wave, it is found that they are exactly synchronous. This is strongly confirmatory of the theory just advanced that this systolic wave in the veins is due to the pressure of the distended first part of the aorta upon the superior vena cava. The alternative is that this wave is due either to a slight amount of tricuspid regurgitation, or to the pressure upon the column of blood in the veins by the sudden bulging of the tricuspid valve on the commencement of the systole. Against these theories it can be pointed out that there is no sign of any filling-up of the vein until after the blood begins to enter the aorta and the ventricle to diminish in volume. Now the valves will begin to bulge towards the veins as soon as the pressure begins to come on them. Also with regard to the regurgitation theory, the regurgitation is almost certain to continue throughout the greater part of the systole, whereas the agency producing this venous wave reaches its maximum at the time of the maximum distension of the first part of the aorta and then rapidly subsides towards the end of the systole. This systolic rise in the vein in this case is a very prominent feature, occurring, as it does, just when the vein is at its emptiest. It here forms the highest point in the venous tracing.

From this the venous tracing falls again until the shoulder occurs which marks the closure of the semilunar valves and the occurrence of the second sound of the heart. This is again followed by a further fall, marking the emptying of the auricle by the ventricular aspiration, developed during its expansion phase.

It may, perhaps, be thought that some or even many of the deductions I have made from these tracings rest upon an insecure foundation, to say the least of it. In answer to this criticism, I can only say that the more I study graphic records of the heart, the more reliable do I find them to be. The fact that the principles of cardiac action, here advocated, do suffice to explain so perfectly the phenomena of cardiac action, as shown in the venous and ventricular tracings, is a strong proof of their accuracy.

SUMMARY OF ARGUMENTS AND FACTS BROUGHT FORWARD IN THE PRECEDING PAGES

Figures in black type refer to important references.
Figures in small type refer to ordinary references.
Figures in parentheses (8) refer to references to clinical cases.

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH DISTENSIBILITY OF THE HEART WALL.

WHAT FACTORS DETERMINE THE DISTENSIBILITY OF THE HEART ?—It is evident, clinically, that the ease with which the heart wall is stretched varies very much according to the age and the individuality of the patient. An amount of strain that will cause dilatation of the heart at one time of life will not do so at another, and the same is true of different individuals at the same time of life.

The estimation of the amount of distensibility possessed by any individual heart is therefore of importance when estimating the amount of its failure when overstrained in any way, p. **34**.

This quality of distensibility is also of value for classification.

Upon what does the heart's power of resisting dilatation depend ?—p. **6**.

It may be upon the muscular strength of its walls. It may depend upon the strength of the fibrous tissues of the heart and pericardium.

It is possible to ascertain the degree in which each of these two factors is concerned in a case of heart failure.

The muscular strength of the heart walls is not the same for all parts of the heart.

The muscular wall of the left ventricle is evidently stronger than that of the right.

The muscular wall of the right ventricle is stronger in some parts than in others, p. 9.

So far as the *fibrous* strength of the heart is concerned there is no anatomical evidence that one part is stronger than another.

Therefore, when the portions of the heart wall which are muscularly the weakest are the first to give way in the face of overstrain, it is a justifiable clinical deduction to say that the heart was evidently depending upon the strength of *its* muscular wall for its power of resisting dilatation.

When, on the other hand, it is found that the parts of the right ventricle wall which are muscularly the weakest, do *not* dilate more readily than the rest of the ventricle wall, and where we find that the left ventricle, which is muscularly so much stronger than the right, dilates to the same extent as the right and possibly to a greater extent (in the face of overstrain), it is justifiable clinically to say that the heart was depending upon its fibrous strength rather than upon its muscular strength for its power of resisting dilatation.

This theory as to the relative parts played by the muscular and the fibrous strength of the heart wall is confirmed by the way in which what we are supposing to be the fibrous strength of the heart wall—as judged by the type of its dilatation—gradually increases as age advances.

In early life and adolescence, when the fibrous tissues are soft and immature, the heart dilates as if it were dependent upon its muscular walls alone, pp. 4, 10.

As life advances, the relative muscular strength of the wall takes less and less part in determining the type of dilatation, just as might be expected to occur (owing to the increasing strength of fibrous tissues) as age advances, pp. 5, 11, 31, 76.

It can therefore be stated that:—

In adult life, clinical study of the behaviour of the heart when dilating in the face of overstrain shows that it is dependent upon its fibrous strength rather than upon its muscular strength for its power of resisting dilatation, p. 10.

What is the source of its fibrous strength?

1. The fibrous tissues of the pericardium are generally recognised as being of value in aiding the heart to resist dilatation, p. 5.

The fibrous support thus obtained will in part, but not entirely, explain the phenomena observable.

2. There must be fibrous elements in the heart wall itself if the phenomena associated with the lessened distensibility that occurs in later life are to be explained, p. 77.

If, anatomically, there is insufficient fibrous tissue in the heart wall to account for the loss of distensibility that characterises later life, this loss of distensibility must be due to some change in the muscular tissue itself—a change, probably similar in nature to that which, when extreme, is recognisable as fibroid degeneration of the heart muscle, p. 77.

This change—whatever it may actually be—is in this book spoken of as an 'increasing rigidity' of the heart wall or a gradually increasing firmness of 'the fibrous elements' of the heart wall, p. 78.

VARIATION IN THE DISTENSIBILITY OF THE HEART ACCORDING TO THE AGE OF THE PATIENT

I. DISTENSIBILITY OF THE HEART IN ADOLESCENCE.—In adolescence the fibrous elements of the heart wall and pericardium have little power (owing to their softness and immaturity) of controlling the distensibility of the heart, which is therefore liable to be great, pp. 7, 10.

Case illustrating this, pp. (7), (8).

The dependence of the heart upon its muscular tissue alone for its power of resisting dilatation ensures the dilatation of the thinner parts of the heart wall before the thicker ones, pp. 4, 9.

Consequently, in adolescence, the right ventricle is more distensible than the left.

Therefore, in overstrain the right ventricle will be the first to give way, and the left ventricle, will be thus protected by the weakness of the right, pp. 5, 11.

Heart failure will, therefore, show first on the right side whether primarily involving the left or the right ventricle, p. 2.

Also in adolescence, the thinner parts of the right ventricle wall will yield before the stronger parts and thus produce a characteristic type of dilatation, p. 9.

In adolescence, the thinness of the upper part of the anterior wall near the pulmonary valves causes this part of the wall to dilate, in face of overstrain, before the rest, pp. 4, 211.

This dilatation of the right ventricle upwards and to the left may be called THE ADOLESCENT TYPE of dilatation, p. 9.

The circumstances under which this adolescent type of distensibility may occur are:—

1. In adolescence owing to immaturity of fibrous tissues.
2. Personal idiosyncrasy. In some elderly persons the distensibility of the heart resembles that of an adolescent, pp. 32, 33, 82, (83), (85).
3. The fibrous tissues may be softened by disease, and thus lose some of their resisting power—notably so in the case of acute rheumatism, p. 11, 343.

II. DISTENSIBILITY OF THE HEART IN ADULT LIFE.—The gradual consolidation and hardening of the fibrous tissues which accompanies the passing of adolescence into adult life causes a gradual change in the distensibility of the heart, pp. 5, 11, 72, 79.

The distensibility of the two ventricles gradually becomes more nearly equal, and consequently the relative frequence of dilatation of the left ventricle increases, 5, 31, 309.

The special distensibility of the thin part of the anterior wall of the right ventricle is gradually lost, and the adult type of dilatation gradually replaces the adolescent as age advances, pp. 11, 31, 33.

III. DISTENSIBILITY OF THE HEART IN LATER LIFE.—The lessening of distensibility, which characterised the change from adolescent to adult life, is still more marked as age advances; and in later life the heart, as a rule, loses a good deal of its distensibility and becomes what may for convenience be called 'relatively rigid,' pp. 73, 76, 79.

Dilatation, when it occurs, is usually of the adult type, pp. 73, 85.

Exceptionally, as a result of personal idiosyncrasy, *dilatation may be of the adolescent type*, pp. 82, (83), (85).

Sometimes the increase in rigidity of the fibrous elements may be so great that the ordinary force of the ventricular contraction is insufficient to cause dilatation of the heart walls.

A clinical group must therefore be recognised where heart failure *without enlargement* of the heart occurs in hearts *of good muscular strength*, but whose walls are abnormally rigid, pp. 73, **86**, (87), (89).

The lessened distensibility has the effect of lessening the frequency with which dilatation of the heart occurs in cases of overstrain of all sorts in later life, pp. 73, 79, 85.

It also lessens the amount of dilatation that will result from any definite degree of overstrain—in comparison with the more distensible heart of a younger individual (pp. 73, 87).

The estimation of the degree of rigidity of the heart wall is therefore of great importance in cardiac diagnosis in later life, pp. 34, **247**.

DISTENSIBILITY IN RELATION TO HEART FAILURE WITHOUT ENLARGEMENT

The distensibility of the heart must also be considered in relation to the cases of heart failure without dilatation which occur where there is great myocardial weakness.

The clinical phenomena observable in cases of myocardial weakness are very different in early life (when the heart walls are distensible) from what they are in later life when they are more rigid.

In the first two or three decades of life a diminution in the size of the cardiac dullness and in the distinctness of its impulse, together with evidences of a diminution in the volume of the blood in circulation, are observable, pp. 50, **54**, 186.

The extent of these changes is proportional to the degree of cardiac weakness present, and in extreme cases there may be not only complete absence of all cardiac impulse, but also complete absence of all signs of cardiac dullness as well. The full gastric resonance will, in such a case, come up to meet the full lung resonance somewhere about the fourth left interspace, giving evidence of great diminution in the volume of the intrathoracic contents. The deficiency must be due to a diminution in the amount of blood in the lungs, heart, &c., pp. **50**, 56, (119), 179.

The causal relationship between the heart weakness and the clinical evidences of its smallness is confirmed by the gradual return of the heart to its normal size as strength returns, pp. **63**, 64, (68), (196).

The clinical phenomena here spoken of cannot be due to a diminution in the actual bulk of the cardiac tissue, but only to the degree to which the heart is filled. The term 'smallness' only refers to the heart when full. The heart when at its emptiest must be the same size as normal. As, however, the heart does not remain empty for much more than $\frac{1}{10}$ of a second (*vide* p. 386), the heart which we percuss clinically is the full heart.

In the distensible heart of adolescence, we are justified in saying that the heart diminishes in size proportionally to the amount of myocardial weakness present, p. 81.

In the less distensible heart of adult life, the diminution in the size of the heart in cases of myocardial weakness is less extreme and may be comparatively slight, pp. 81, (158).

In adult and later life, valuable evidence of dangerous myocardial weakness may be given by the failure of a heart to dilate under circumstances which, considering its degree of distensibility, ought to have caused dilatation, pp. (64), 71.

Cases of pneumonia where the absence of cardiac dilatation showed dangerous myocardial weakness, pp. (97), (105).

In the relatively rigid heart of later life, there is often little or no certain evidence of any diminution in the size of the heart in cases of extreme weakness. In such cases it is justifiable to deduce that the degree of rigidity present prevents any lessening in the size of the very weak heart just as the presence of rigidity in a fairly strong heart may prevent the occurrence of dilatation when heart failure from overstrain occurs, pp. 74, 79, 91.

Therefore, in the more rigid heart of later life, signs of myocardial weakness must be looked for in connection with compensatory diminution in the amount of blood in circulation rather than in connection with changes in the heart itself, pp. 57, 94.

Clinical Estimation of the Distensibility of the Heart.—It is evident that we cannot estimate correctly the clinical significance of the *presence* or of the *absence* of alterations in the size of the heart in cases of heart failure, unless some estimate can at the same time be made of the amount of distensibility of the heart wall, p. 34.

In Early Adolescence, the estimation of the heart's distensibility is easy, owing to the ready response of the heart wall, either by enlargement or by diminution in size, to alterations in the mode of action of the heart, p. 10.

In a moderately strong heart, the distensibility is shown by the readiness with which the heart dilates and by the extent of the dilatation when overstrained, and in a very weak heart by the readiness with which the cardiac dullness diminishes in size and by the extent of that diminution, pp. 10, 52, 54.

In Late Adolescence, a slight lessening in the heart's distensibility will be shown by the occurrence of some dilatation of the right ventricle towards the right (in moderate degrees of overstrain), i.e. by a tendency towards the adult type of dilatation, p. 33.

In Adult and Later Life—I. In hearts of good or moderate strength (as judged by the cardiac sounds and impulse, and the jugular bulb pulsation, &c.).

1. Considerable distensibility will be shown by the occurrence (when the heart is overstrained) of a combination of the the adolescent and the adult types of dilatation of the right ventricle.

A greater preponderance of the adolescent type will show a higher degree of distensibility, and vice versa, pp. 11, 33.

A preponderance of right-sided dilatation, as compared with left-sided, will also show a high degree of distensibility, p. 11.

2. A less degree of distensibility will be shown when the adult type alone occurs; and the more dilatation of the left ventricle in overstrain preponderates over that of the right the less must the heart's distensibility be, p. 11.

Also the less the amount of dilatation that occurs with any definite degree of overstrain the greater must the relative 'rigidity' of the heart be, p. 86.

3. If cardiac failure be unaccompanied by dilatation it shows a great lessening in the distensibility of the heart (i.e. a considerable amount of 'rigidity'). The degree of rigidity is measured by the amount of cardiac overaction observable, pp. **86**, 148.

(It can safely be asserted that a heart which shows 'failure without enlargement' in spite of a considerable amount of overaction is giving more evidence of rigidity of its walls than one which shows 'failure without enlargement' in combination with a less amount of overaction, p. 8.)

Abnormal rigidity of the heart wall can be diagnosed when the heart fails to dilate under circumstances that might reasonably be expected to lead to dilatation, pp. **73**, (87), (89).

Evidences of increased right ventricle activity are of value in estimating the amount of cardiac rigidity in later life, p. 86.

Diagnostic value of seeing the relationship of the strength of the venous pulse in the jugular bulb to the amount of dilatation, pp. (87), 97, **137**.

II. In hearts of considerable muscular weakness (as judged by the cardiac sounds and impulse and the evidences of compensatory reduction in the volume of blood in circulation).

1. The presence of some degree of 'rigidity' (i.e. loss of distensibility) will be shown by the fact that the *diminution* in the size of the cardiac dullness is not so great as would be noticeable in the heart of an adolescent with the same amount of cardiac weakness, p. 96.

2. A higher degree of 'rigidity' will be shown by the fact that there is no reduction at all in the size of the heart.

The greater the degree of weakness which is present the stronger will the evidence of 'rigidity' be, pp. 91, 112, **150**.

3. Where a heart shows no diminution in its size in spite of considerable weakness and it is therefore difficult to estimate the

actual amount of its distensibility, it is nevertheless sometimes possible to do so, *on its recovery*, by noting the occurrence of ' dilatation as a sign of returning strength.' For this purpose the size of the right ventricle, as shown by the amount of dullness in the third left interspace, and in the fourth and fifth left and right interspaces near the sternum, must be very carefully watched, p. 63.

4. Often in later life it is not possible (when the heart is relatively rigid) to estimate with any certainty the degree of muscular weakness that is present (owing to the uncertainty as to the indications given by the pulse and the blood pressure, if normal or higher than normal, p. 143, and to the interference, due to tissue rigidity, with the indications that would be given otherwise by compensatory lessening of the blood volume, p. 72.

In such a case no estimate of the rigidity may be possible, p. 148.

5. The presence of *symptoms* of heart weakness unaccompanied by diminution in the size of the heart and without evidence of a compensatory reduction in the blood volume points to the presence of a dangerous amount of rigidity of the heart walls.

Diagnostic points in very weak hearts, p. 147.

Very weak heart showing distensibility in later life, pp. (157), (159).

Post-mortem estimate of the distensibility of the heart.—This is possible in early life, to some extent. In later life the changes due to rigor mortis would probably interfere with any useful estimate being made, p. 81.

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH HEART FAILURE WITH ENLARGEMENT

FACTORS WHICH DETERMINE THE TYPE AND THE AMOUNT OF CARDIAC DILATATION

The question of dilatation of the heart is a simple one.

If the intraventricular pressure developed by the muscular wall exceeds the resisting power of the weakest part of the wall, that part will be stretched and heart failure with dilatation will result, pp. 6, 9, 31.

If intraventricular pressure, in excess of the resisting power of the ventricle walls, cannot be developed, no dilatation of the ventricles can occur and heart failure *without* enlargement will result, pp. 6, **54, 86, 91.**

In other words, dilatation results when the force developed by the stronger portions of the ventricle wall causes over-distension of the weaker parts, pp. 6, 31, 211.

Which part of the heart wall is the weakest ?

For its power of resisting dilatation the heart depends on two factors :—

1. The resisting power of the fibrous elements of its walls —especially the pericardium, both visceral and parietal, pp. 5, 31.
2. The tonic strength of its muscular walls, pp. 4, 9.

The fibrous tissues gain in strength and resisting power with maturity, and upon them the heart mainly depends in adult life for its power of resisting dilatation, pp. 5, 11, **31**, 33, **76.**

The fibrous tissues continue to increase in strength and resisting power, as a rule, as age advances.

The amount and the type of cardiac dilatation may therefore be expected gradually to change as age advances.

The heart in later life has more 'mechanical' (as distinct from muscular) resisting power. It has less 'distensibility' and more 'rigidity,' pp. 73, 76.

Dilatation is less frequently a result of overstrain than was the case earlier in life, pp. 31, 71, 73, **76,** 87.

VARIOUS TYPES OF DILATATION

1. IN ADULT LIFE, when the fibrous tissues have good resisting power, in the case of both right and left ventricles, the apical portion seems clinically somewhat stronger than the basal part, and therefore the relative *muscular* thinness of this latter portion must take *some* share in determining where dilatation will occur, p. 31.

Dilatation, when it occurs, mainly involves the part of the ventricle wall which is adjacent to the auriclo-ventricular valve, pp. 5, 10.

In the case of the left ventricle this causes a widening of the base of the ventricle towards the left, and in the case of the right ventricle a widening towards the right, pp. 47, 40.

This constitutes what may be called the adult type of dilatation, pp. 11, 31, &c.

The thinness of this basal portion has a most important action in safeguarding the ventricle.

Its dilatation, when it reaches a certain point, so affects the auriculo-ventricular orifice as to cause incompetence of the valve and escape of some of the ventricular contents into the auricle, p. 32.

Were it not for this well-known safety-valve action, dilatation of the ventricle, having once started, would rapidly increase till the walls either gave way or became too thin to maintain adequate blood pressure, p. 32.

IN LATER LIFE, when the fibrous tissues are more resistant still, there may be so much gain in the resisting power of the fibrous elements that these thinner portions of the heart walls lose their relative distensibility. When this takes place no dilatation of the ventricle can occur (owing to the inadequacy of such force as it can develop), and then any enlargement would be confined to the auricles and great veins, p. 86.

2. ADOLESCENT TYPE OF DILATATION.—In the early decades of life, on the other hand, when, owing to immaturity or other cause, the fibrous elements of the heart wall and pericardium have little or no resisting power, the heart will

dilate as if it were dependent upon the strength of its muscular walls alone, pp. 4, **9**.

Under these circumstances in the case of the LEFT VENTRICLE, there is no evidence that the *type* of dilatation is materially different from that occurring when the fibrous elements have good resisting power.

In the case of the RIGHT VENTRICLE, however, there is a well-marked departure from the type just described; for certain parts of the ventricle wall are decidedly weaker muscularly than the rest, and these are the first to give way in the face of any abnormal strain or when for any cause the muscle is abnormally weak, pp. 4, **9**.

The weakest parts are the conus arteriosus and the adjacent part of the anterior wall of the ventricle, p. **9**.

The strongest parts are the interventricular septum and the apical portion of the ventricle, in consequence of its muscular bands.

Intermediate in strength is the part adjacent to the auriculo-ventricular groove and the remainder of the anterior wall.

Also, in consequence of the relative muscular weakness of the right ventricle as compared with the left, dilatation is, usually, confined to the right ventricle alone, unless the heart is severely overstrained, pp. 5, 46, 308.

When may this type of dilatation be expected to occur? In early life, up to and including adolescence, the softness and immaturity of the fibrous elements will so greatly lessen their resisting power as to make the heart dependent, almost entirely, upon its muscular strength for its power of resisting dilatation. In adolescence, therefore, the thin part of the anterior wall which is adjacent to the pulmonary valves will be the first to yield in the face of overstrain, and the resulting type of dilatation may well be called the ADOLESCENT TYPE, pp. 5, **9**, 330, &c.

Adult and adolescent types of dilatation compared, pp. 5, 33, 330.

The adolescent type of dilatation will also occur apart from 'absolute' overstrain, whenever the muscular strength of the wall is lessened from malnutrition or other cause. These may be called cases of 'relative' overstrain. For instance, in cases of simple anæmia, where there is no overaction of the right ventricle, or where the heart is recovering from myocardial weakness, pp. **63,** (200), (304).

The adolescent type of dilatation will also be found in adult life wherever the resisting power of the fibrous elements of the heart wall is less than normal in consequence of disease—such as acute rheumatism—or in consequence of personal idiosyncrasy, pp. 11, **82,** 343.

Adolescent type of dilatation in later life after the age of fifty, pp. (83), (85).

The amount and the type of dilatation found as age advances will be determined by the degree of maturity attained by the fibrous tissues, and the adolescent type will year by year gradually change into the adult type, pp. 5, 33.

3. INTERMEDIATE TYPES OF DILATATION. — The above two types of dilatation only occur, as a rule, in late adult life and in early adolescence, respectively. In the intermediate period of life, intermediate types usually occur, p. 33.

APPORTIONMENT OF STRAIN BETWEEN THE RIGHT AND LEFT VENTRICLES

In the first place the word 'strain' needs definition; for a normally strong heart may be overstrained through excess of work—this is a condition of 'absolute overstrain.' Or the overstrain may result from the heart being too weak to do an amount of work that does not exceed its normal limits—a condition of 'relative overstrain,' pp. 310, 342.

CONDITIONS CAUSING OVERSTRAIN

1. Ordinary physical exertion, pp. (310), (316).
2. Anything interfering with the pulmonary circulation—such as pneumonia, or the simple anæmia of adolescence, pp. 245, 343, (360).
3. The extra work thrown upon the right heart by valvular disease of the left ventricle, p. 343.

Amongst the causes of relative overstrain all causes of cardiac weakness would have to be mentioned, and prominently amongst them—so far as clinical importance is concerned—the weakening of the heart muscle due to such infective conditions as influenza and diphtheria, and the softening and weakening of the heart which results from rheumatism, pp. 11, **343**.

As regards the apportionment of strain between the two ventricles, the last two causes of absolute overstrain involve the right ventricle only.

As regards the strain of physical over-exertion.

In adolescence the weakness of the right ventricle so protects the left that it is not easy to say that any particular type of strain involves one ventricle more than the other, pp. **46**, **309**.

Under adolescent conditions, therefore, signs of overstrain must be looked for as a rule in the right ventricle, even though the strain be more likely to involve the left side, pp. 11, **308**.

Under adult conditions the resisting power of the two ventricles being more nearly equal, overstrain usually involves the left ventricle as well as the right, pp. **308**, **316**.

Careful observation as to the effects of different types of over-exertion might show that some kinds involve one ventrical more than the other, p. 310.

But the inaccessibility of the left ventricle would prevent slight signs of failure being noticeable, pp. 2, 47.

Also the structural arrangements of the left ventricle ensure that any ineffective action on its part immediately causes embarrassment of the right, and so tends to divide the strain between the two sides of the heart, p. 2.

PATHOLOGICAL ASPECTS OF DILATATION OF THE RIGHT VENTRICLE IN ADOLESCENCE AND EARLY ADULT LIFE, p. 247

Enlargement up and to the left, pp. 233, 247.

Change in level of pulmonary valves caused by upward dilatation ; sometimes under first rib, pp. (233), (251).

1. Changes in pulmonary artery, p. **251**.

It is shortened, thus causing relaxation of its elastic walls, pp. (233), 252, **262**.

It is often displaced as much as one inch to the left of its normal position, *vide* figs. **60**, 61, p. 248.

It is brought nearer to the anterior chest wall.

Its direction in relation to the axis of the right ventricle is altered, pp. 252, 336.

It is liable to undergo temporary aneurysmal dilatation under normal or increased systolic blood pressure, p. 253.

Its relationship to the aortic arch is altered, pp. 215, 259.

Enlargement to the right is not present in heart failure of adolescence unless dilatation be extreme, pp. 238, 354.

Position of the right auriculo-ventricular groove in adolescent dilatation, and in dilatation towards the right, pp. 41, (236).

Dilatation of the right auricle does not usually cause dullness or pulsation observable clinically, p. 41.

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH THE CLINICAL ASPECTS OF HEART FAILURE WITH ENLARGEMENT

WHEN THE HEART IS DISTENSIBLE OR MODERATELY DISTENSIBLE.—Early signs of heart failure show first in the right ventricle for anatomical and physiological reasons, p. 2.

In the distensible heart of adolescence the relative weakness of the right side so saves the left, that evidences of heart failure, unless it be extreme, are practically limited to the right ventricle, pp. 11, 46, 309.

In the extreme heart failure, due to valvular disease of the left ventricle, phenomena, usually associated with the adult type of dilatation, may be superadded to those characteristic of heart failure in adolescence, pp. 30, (339).

ADOLESCENT OR UPWARD TYPE OF DILATATION OF THE RIGHT VENTRICLE

Symptoms, pp. 12, 342.

Breathlessness. Relief by a ‘ deep breath,’ pp. 14, 22.

Pain and tenderness. Globus hystericus, pp. 14, 212, 342.

Physical signs, pp. 17, 330.

Veins of neck ; fullness and false pulsation, pp. 14, 137, 332.

Inspection of the heart, pp. 17, 331.

Increased cardiac pulsation in third and second interspaces, pp. 17, 331.

Character of the impulse on inspection, pp. 17, 331.

Greater at end of inspiration, pp. 18, 232.

Passage of wave of contraction down and out, p. 18.

PALPATION of the heart in the adolescent type of dilatation.

Situation of apex, often in fourth interspace, pp. 19, **234**, 237, 332, **349**.

Great increase in area of cardiac pulsation in well-marked case, p. (306).

Diagnostic value of pulsation felt in the third left interspace in and external to the nipple line, p. 19.

Shock due to the closure of the pulmonary valves, often felt in second interspace and sometimes some distance from the sternum when overstrain is severe, as in the heart failure of anæmia, pp. 232, (286), 332.

Presence of a systolic thrill over pulmonary artery in anæmia, pp. 19, 255, 332.

Thrill conducted to arteries of neck, especially left carotid, pp. 216, 259, (291).

No increase to right of sternum in adolescent type of dilatation, pp. (236), 238, 354.

Upward dilatation of ventricle implies the absence of tricuspid regurgitation unless heart failure be extreme, pp. 211, **239**, 269.

PERCUSSION of the heart in adolescent type of dilatation, pp. 25, 332.

Method of percussion. Value of both strong and light percussion, p. 20.

Increase of cardiac dullness upwards and to the left, pp. 25, 211, **228**.

Usually both upwards and outwards, p. 289.

Sometimes mainly upwards, p. 290.

Sometimes involves only the conus arteriosus, pp. 292, (293), (294).

Extra strain on the conus arteriosus, p. 10.

Dilatation of conus alone is a sign of myocardial weakness, p. 293.

Sometimes dilatation is mainly outwards to the left, p. 298.

Diversity of type in the overstrain in anæmia, p. 289.

No dilatation to the right in ordinary type of adolescent dilatation from overstrain, pp. 238, 354.

Extreme dilatation to the right may be superadded to this type in severe valvular disease in adolescence, p. (339).

Age as a factor in determining the type of upward dilatation, pp. 290, 349.

Lower border of the cardiac dullness often raised by rise of the diaphragm, as shown by the level of the gastric resonance, pp. 181, 289.

Rise of diaphragm in relation to the shape of the cardiac dullness, pp. 246, 289.

Lower border of heart, as judged by position of apex and area of pulsation, may be raised apart from rise of diaphragm.

High diaphragm in adolescent dilatation of the right ventricle, explainable in other cases by malnutrition or other cause of high diaphragm, p. 163.

Two cases showing high apex with and without rise of diaphragm respectively, pp. (290), (292).

Frequency of upward displacement of apex in adolescent heart failure of anæmia, pp. 234, 349.

Statistics as to the frequency of the various phenomena associated with it, pp. 349 to 356.

Displacement upwards of apex due to its upward rotation by a slight general stretching of the anterior wall of the *right* ventricle towards the *left*, pp. 237, 357.

Upward displacement of the apex more common in patients over nineteen years of age and associated with a diminution in the extreme distensibility of the upper part of the right ventricle wall, p. 349.

Increase of cardiac dullness upwards with recumbency, pp. 266, (298), (305); sometimes associated with a corresponding rise in the level of the heart apex, p. (313); sometimes due entirely to the lessened tension of the anterior wall of the ventricle when it does not aid in supporting the weight of the heart, and without any rise in the apex, p. (311).

Increase in the amount of dullness in the second and third interspaces with slight exertion or cardiac excitement, pp. 8, (268),

Bearing of this increase in dullness upon the cause of the pulmonary systolic murmur, pp. 257, 268.

AUSCULTATION of the veins and arteries in the adolescent type of dilatation, i.e. in 'heart failure with enlargement in distensible hearts.'

Auscultation of the veins.

Presence of a 'bruit de diable,' especially in the heart failure of anæmia, pp. **15,** 337.

Its characters; louder during diastole; disappearance with recumbency, pp. 16, (285), 338; causation at points of constriction of veins, pp. 15, 337.

Auscultation of arteries, p. 226.

Frequency of an arterial compression murmur in the subclavians in the adolescent dilatation of anæmia, pp. 226, 281, (299)

Conduction of pulmonary systolic murmur into the arteries of the neck, especially the left carotid, pp. 215, **259,** 335.

AUSCULTATION OF THE HEART

PULMONARY SECOND SOUND.—Importance of noting the loudness of the pulmonary second sound as giving a measure of the amount of extra work that the right ventricle is doing—i.e., its degree of overstrain and the efficacy of its muscular response to its increased load, pp. 29, 42, 162, 337.

ACCENTUATION of pulmonary second sound in anæmic heart failure, pp. **244,** 337, 415, 421.

The extremely loud second sound in anæmic heart failure is apparently due to overstrain caused by some interference with the pulmonary circulation in anæmia, because there are no evidences of left ventricular overstrain, p. 245. The left ventricle having, apparently, less work to do than normal, p. 240.

The cause of hindrance to the pulmonary circulation is uncertain, pp. 246, 343.

Pulmonary second sound in anæmia is louder than when the right ventricular failure is secondary to left ventricular failure in valvular disease, pp. 244, 415, 421.

Need for making allowance for the fact that in adolescent dilatation the pulmonary valves are closer to the anterior chest wall than normal.

REDUPLICATION of the second sound.

Importance of noting whether the pulmonary half precedes the aortic, or vice versa.

In ordinary overstrain in adolescence, and especially in anæmia, the pulmonary half precedes the aortic, pp. **244**, (297).

When right ventricular failure, with an accentuated pulmonary second sound with reduplication is (say) secondary to left ventricular failure, the result of high arterial resistance, it may sometimes be noted that the aortic half precedes the pulmonary, thus giving an important indication for treatment.

In connection with the effect which respiration is known to have on the action of the right ventricle, it is of interest to note that careful measurements of the loudness of the pulmonary second sound shows that the second sound occurring with the first cardiac beat after the commencement of expiration is often measurably louder than the rest, p. **232**.

Loudness of the right ventricle first sound, p. **244**.

It is important to note the loudness of the first sound heard over the right ventricle in adolescent heart failure, although there are other indications of greater value. In later life its diagnostic value is great.

PULMONARY ARTERY SYSTOLIC MURMUR, p. 254

A most valuable sign of adolescent heart failure, pp. **26**, **215**, **254**.

Its characters. WHAT is it?

An aneurysm-like murmur (sometimes accompanied by an aneurysmal thrill), pp. **255**, **334**.

It accompanies, but does not replace the first sound of the heart, because even when the murmur is loud the first sound usually can be heard quite pure over the lower end of the sternum, pp. **257**, **259**.

It has rather a low tone as a rule—as compared with a mitral systolic murmur, for instance, p. **255**.

It is a very variable murmur, p. **256**.

It varies very greatly with change of position, being often quite loud when the patient is recumbent and faint or absent when erect, pp. **256**, **266**.

It is always increased with recumbency, pp. 256, 334.

It is greatly increased in loudness by slight exertion, p. 257.

It is greatly increased in loudness by excited heart action, pp. 257, (268).

It can sometimes be noticed to vary in loudness with respiration, just as is the case with the pulmonary second sound.

It varies according to the strength of the heart rather more than an ordinary valvular murmur. It may be faint when the heart is weak, and rapidly gain in loudness in a few days as the heart's strength improves, p. 263.

In the same way a very loud murmur may very rapidly lessen in loudness in a week or two on the improvement of the heart, fig. 75, p. 261.

When is the murmur audible?

It is systolic in rhythm and apparently accompanies the first sound of the heart. If accurate measurement were possible it would probably be found to commence just after the closure of the auriculo-ventricular valves and attain its maximum when the pulmonary artery reaches its fullest (i.e. at the time when the recoil wave, due to its elasticity, returns into the ventricle), p. 257.

Where is the murmur audible?

Its point of maximal intensity (its P.M.I.) appears to coincide with the situation of the pulmonary artery, p. 258.

In slight cases of adolescent dilatation this may be in about the normal situation at the sternal end of the second left interspace, p. 260.

Sometimes in extreme cases the artery (and the P.M.I. of the murmur) may be 1 or 1½ inches away from the sternum, p. 258.

The situation of the pulmonary artery may sometimes be shown by the shock due to the closure of the semilunar valves being palpable, pp. 232, (286), 332.

The P.M.I. of the murmur may sometimes be noticed to change a little with change of posture—as is the case with the P.M.I. of the pulmonary second sound (due to the increase in the amount of upward dilatation which recumbency causes), p. 258.

Its direction of conduction, pp. 215, 259, 335.

When moderately loud it is heard over an area a few inches across, spreading upwards and outwards from its P.M.I., p. 260.

Not heard below the fourth rib. (When apparently heard lower down this is really an independent tricuspid murmur), pp. 259, (278).

Not well heard to the right of the sternum unless very loud. (When apparently conducted to the right this is really an independent superior vena cava murmur.)

Sometimes conducted up into the neck and then is usually better heard in the left carotid than the right. (This is due to the close contact of the dilated pulmonary artery, in which the murmur arises, with the second part of the arch of the aorta, pp. 215, 259, 335.

How is the pulmonary systolic murmur caused ?

The clinical evidence shows that it is a murmur of right ventricular origin, pp. 26, **263.**

That it does not arise at the tricuspid valve, pp. 259, 263.

That its clinical features all suggest the pulmonary artery as its point of origin.

THEORY as to its mode of origin in the pulmonary artery. The murmur results from the pulmonary artery losing its normal cylindrical form and becoming more or less spherical when the elastic tissue which constitutes its wall is inadequate to resist the blood pressure developed within the artery by the right ventricle, pp. 270, 335.

In other words, the murmur arises in a temporary aneurysm-like dilatation of the pulmonary artery, pp. 26, 252, 336.

Such a dilatation can arise—

1. Simply from an excess of blood pressure in an artery whose elastic wall is practically normal, p. 273.

This rarely happens. Records of such a case, pp. (274) and (300).

2. From relaxation of the arterial wall lessening its resisting power. This is the ordinary cause of the murmur, pp. 26, 272.

3. This theory necessitates a blood pressure adequate to cause dilatation of the artery, pp. 27, 272.

4. A pulmonary orifice smaller in size than that of the

vessel when dilated; otherwise the pulmonary artery could not constitute an aneurysmal dilatation, but only a cylindrical prolongation of the cavity of the conus arteriosus in which no murmur would arise, pp. 27, 272, 275.

5. In addition to the above-mentioned factors, the formation of murmur-producing eddies in the dilated artery is facilitated by the fact that the upward dilatation of the right ventricle, which causes the murmur, results in an alteration in the axis of the pulmonary artery as compared with that of the ventricle, the blood being projected against its anterior wall and not directly into its lumen as in the normal heart, pp. 252, 271, 336.

The ordinary phenomena already described are easily explained by the variations in the blood pressure and by the variations in the degree of relaxation of the elastic wall of the pulmonary artery.

A lessening in the loudness of the murmur, owing to dilatation of the pulmonary orifice, is seen in cases of extreme adolescent dilatation, pp. 264, 275.

Variations in the murmur due to any variation in the direction of the pulmonary artery are neither likely to occur nor likely to be of clinical importance.

THE CLINICAL CHARACTERISTICS of the pulmonary systolic murmur harmonise with the above theory.

The murmur is of an aneurysmal type, p. 255.

The extreme and rapid variability of the murmur suggests its production in an elastic structure—such as the pulmonary artery—which can readily recover itself on the removal of a disturbing factor, p. 263.

VARIATIONS IN THE AMOUNT OF RELAXATION of the pulmonary artery.

The conditions resulting from upward dilatation of the right ventricle are such as to produce an amount of relaxation adequate to give rise to a murmur, pp. 251, 336.

The raising of the level of the pulmonary valves by the upward dilatation of the ventricle will approximate the commencement of the pulmonary artery to its termination at its bifurcation—the latter being a fixed point. The arterial wall will thereby be relaxed, p. 249.

The relaxation will be directly proportional to the amount of upward dilatation, and if the other variable factors are constant the loudness of the murmur ought to be proportional to the amount of upward dilatation, pp. 26, 264.

This is found to be the case clinically; for the loudness of the murmur and the amount of upward dilatation are both increased by :—

1. Change of posture; recumbency causing a marked increase, pp. 27, 266.
2. Excited action of the heart, pp. 228, 257, 268.
3. Slight amounts of exertion, p. 268.
4. Also in a series of cases (if those be excluded where there is a likelihood that the pulmonary orifice is becoming dilated), the loudness of the pulmonary systolic murmur is found to be proportional to the amount of upward dilatation present, p. 264.

VARIATION IN THE BLOOD PRESSURE as a factor in the causation of the murmur also confirms the above theory.

The striking variations in the loudness of this murmur are due to the fact that any sudden increase of blood pressure—such as results from slight exertion or excited action of the heart from emotional or other causes—not only increases the distensile pressure which the right ventricle develops in the pulmonary artery at each systole, but also (owing to the distensibility of the upper part of the ventricle wall in adolescence) such increase in the heart's activity also increases the amount of upward dilatation, and therefore the degree of relaxation of the artery. Increased cardiac activity therefore, has the double effect of increasing both the relaxation of the arterial wall and the distensile force, pp. 268, 272.

Presence of accentuation of pulmonary second sound of value in estimating the strength of the right ventricle, pp. 244, 269.

When the heart muscle is too weak—as in severe cases of simple anæmia—to develop an adequate distensile force, a considerable amount of upward dilatation may be accompanied by but a faint murmur, p. 273.

In such a case, return of cardiac strength will be accompanied by increased loudness of the murmur, and it will then become proportional to the amount of dilatation, p. (305).

In such a case as this, the return of cardiac strength will often be accompanied by a decided increase in the amount of dilatation, pp. (294), (297), (304).

Briefly, it can be said that with a constant blood pressure in the artery the loudness of the murmur will be proportional to the degree of relaxation caused by the upward dilatation of the ventricle, or with a constant degree of relaxation of the vessel wall the loudness of the murmur will be directly proportional to the amount of the blood pressure.

Variation in loudness of murmur with varying cardiac strength, p. (304).

In confirmation of this it may be noticed that in the exceptional cases where the murmur is not due to relaxation of the arterial wall or where the anterior wall is sufficiently strong for its amount of dilatation not to be lessened in the erect attitude, the slight increase in cardiac activity which the erect attitude calls out, is sufficient to make the pulmonary murmur louder in the erect than in the recumbent attitude, pp. (274) and (300).

Cases where the murmur was not increased on lying down, pp. (300), (302), (303).

Accentuation of pulmonary second sound lessened on lying down, p. 300.

VARIATIONS IN THE SIZE OF THE PULMONARY ORIFICE

For the production of murmur-producing eddies it is necessary that the orifice of entrance be smaller than the chamber in which the eddies are to be produced, p. 271.

It may therefore be stated that, other factors being constant, the loudness of the pulmonary murmur will be proportional to the smallness of the pulmonary orifice in comparison with the size of the dilated artery, and conversely when the size of the pulmonary orifice approaches that of the dilated pulmonary artery the production of a murmur is no longer possible.

In a series of cases where there is very extreme upward dilatation of the right ventricle it is found that the pulmonary artery murmur is often faint or even quite absent, pp. 264, 275, (306), (314).

Lessening of pulmonary murmur with increase of heart vigour and of dilatation, pp. (294), (304).

In further confirmation of this theory, it is sometimes found that when once the pulmonary murmur has disappeared (owing to the stretching of the pulmonary orifice), it does not reappear when the upward dilatation of the ventricle lessens. In such a case, we shall find the murmur absent, in spite of there being a moderate amount of upward dilatation—such an amount as is usually accompanied by a loud murmur, p. 297.

Another possible factor must be referred to—namely, that in cases of extreme dilatation the support given by surrounding structures may to some extent lessen the aneurysmal dilatation of the pulmonary artery, pp. 27, 276.

OTHER AUSCULTATORY PHENOMENA sometimes found in the adolescent type of dilatation.

A third sound of the heart, audible over the right ventricle may be heard, pp. 282, 340.

Position of interventricular septum shown by the area of audition of the third sound, pp. 284, 341.

Effect of change of position on the third sound, p. 284.

Third sound only audible when recumbent, p. (313).

Mid-diastolic tricuspid murmur audible in right ventricular dilatation—especially in anæmic heart failure, pp. 282, (283), 341.

Arterial compression murmur (especially in anæmia), pp. 226, 281, (299).

Tricuspid systolic murmur infrequent in anæmia, p. 239.

Sequence of clinical phenomena observable in gradually increasing degrees of cardiac failure, pp. 28 to 30.

CARDIAC FAILURE WITH ENLARGEMENT IN MODERATELY DISTENSIBLE HEARTS, I.E. ADULT TYPE OF DILATATION, p. 47

The CLINICAL PHENOMENA are those usually described as characterising heart failure.

Symptoms, p. 36.

Breathlessness more important in adult life than adolescence, p. 36.

Pain, p. 37. Greater liability to cardiac pain in adult than adolescent.

Other symptoms—cyanosis, dropsy, &c.

PHYSICAL SIGNS relating to the right ventricle.

Inspection. Increase of pulsation just to left of sternum, p. 40.

Later on, pulsation to the right of sternum and in the epigastrium, p. 40.

Fullness and pulsation of the veins of the neck, p. 38.

False pulsation at first, true pulsation later on, pp. 39, 137, 338.

Information from radial pulse. Its unreliability in adult and later life, pp. 35, (35), 143.

Force and volume of the impulse in jugular bulb of great value in estimating the amount of cardiac failure, p. 39, **134**.

Force and volume of systolic pulsation in neck veins (if any) also of importance, p. 135.

Character and position of apex beat to be observed.

Presence of abnormal pulsation just to left of sternum and its force to be noted, pp. 17, 29, 40.

Also extension of area of pulsation to the right or downwards, or in severe cases, pulsation of the liver, p. 40.

Case of right ventricle failure with venous plethora, but no dilatation to the right, fig. 91, p. 278.

Percussion of the heart. Shows the measure of dilatation to the left and to the right with greater exactness than palpation, p. 25.

Auscultation, p. 26, of the veins, pp. 24, 219, (291).

The points to be noted in connection with the heart are:—

Study the relative loudness of right ventricle first sound as compared with the left. Also the loudness of the pulmonary (p. 29) and aortic second sounds. If reduplication be present notice which half of the second sound occurs first, as indicating whether the right heart or the left be more overstrained.

Note the presence or absence of murmurs.

In early heart failure the presence of a pulmonary systolic murmur in early adult life may be noted, p. 26.

Of most importance are the signs of tricuspid regurgitation, p. 8. A third sound and mid-diastolic murmur may occur in right ventricular dilatation, pp. 46, 282.

TRICUSPID REGURGITATION

A sign of heart failure.

Due to dilatation of basal part of ventricle, pp. 32, 37.

 Compensatory effect of it in limiting dilatation, p. 32.

Its effect on right auricle, p. 41.

 Tonic muscular response by wall of auricle, pp. 509, 521, 585.

 Dilatation of auricle, and see 'Auricle' in subject index, p. 210.

 A dilated auricle does not as a rule come near anterior chest wall, p. 41.

Its effect upon the great veins forming a part of the auriculo-venous reservoir.

 Tonic muscular response by their walls, pp. 507, 513, 586.

 Degree of fullness of auriculo-venous reservoir shown by the volume of the pulsation felt in jugular bulb, p. 132.

 Chronic over-distension causing descent of diaphragm, p. 362.

 In other ways, *vide* Summary—'Auriculo-venous Reservoir.'

 Force with which the reservoir is distended judged by the strength of the pulsation in the jugular bulb, pp. 133, 137.

Its relation to the inferior and superior vena cava, pp. 37. 210.

 Importance of the Eustachian valve, p. 38.

Regurgitation downwards into the liver, p. 39.

 Clinical changes in liver which result.

 Enlargement, pp. 39, 210.

 Effect of over-distension upon percussion dullness, pp. 24, 191.

 Increased conductivity to intestinal resonance is caused by venous engorgement, pp. 24, 191.

 Pulsation of the liver, p. 210.

 Pain due to over-distension of the liver, p. 37.

 Ascites.

Regurgitation upwards into the superior vena cava.

Its effect upon the superior vena cava, pp. **44**, **218**.

Its effect upon the veins of the neck, p. **39**.

Fullness and false pulsation when slight, p. **38**.

Fullness and false pulsation when ventricle muscularly weak, p. **137**.

Systolic pulsation in the neck veins, p. **132**.

Rhythm of the pulsation, p. **135**.

Sometimes so forcible as to resemble arterial pulsation, pp. (220), (222).

Its clinical recognition in other ways.

Usually associated with increase of cardiac dullness to the right of the sternum, pp. **40**, (321), (339).

Often with pulsation to the right of the sternum.

Sometimes there is no pulsation either in neck veins or liver, p. **339**.

AUSCULATORY SIGNS.

A SYSTOLIC MURMUR may be produced by tricuspid regurgitation in either or all of three situations, pp. **43**, **217**, **338**.

AT THE TRICUSPID VALVE.

The tricuspid systolic murmur, pp. **43**, **217**, **339**.

Its occurrence. Not always present, p. **43**; absent in spite of dilatation of ventricle and regurgitation, pp. (278), (280), (320).

Similar absence of systolic mitral murmur in case of great dilatation of the left ventricle, pp. (281), (320).

Its character, P.M.I., &c., pp. **43**, **217**, **339**.

Variation with attitude of patient. Usually less loud when erect. Absent. Fig. 73, pp. **261**, (303). No change, p. **301**.

Variation with respiration, p. **500**.

Area of audition, pp. **43**, **339**.

Propagation downwards towards liver, pp. **44**, **218**, **339**.

Propagation upwards into veins, pp. (221), **223**.

Propagation towards the back, p. **445**.

Diagnostic importance of noting relationship of right ventricle first sound to a true tricuspid systolic murmur, pp. **218**, **223**.

IN THE SUPERIOR VENA CAVA.

The superior vena cavà murmur, pp. 44, 218.

Effect of regurgitation upon the superior vena cava. Its dilatation results in a relative constriction by the unyielding fibres of the pericardium where it passes through it, p. 44.

Characters of this murmur.

Somewhat similar to those of the tricuspid murmur, pp. 45, 218.

Point of maximum loudness. Third and second right interspaces, pp. 45, 218.

Conduction into veins of neck, pp. 45, 219.

Diagnosis from a true tricuspid murmur, p. 45.

Diagnosis from an aortic systolic murmur, pp. 45, 218, (222).

Similarity to an aortic systolic murmur, p. 221.

Diagnosis from a pulmonary systolic murmur, *vide* pp. 219 and 334.

Its diagnostic importance—especially in heart failure of later life, pp. 46, 87.

Superior vena cava murmur and no tricuspid murmur, pp. (222), (224), (320).

Superior vena cava murmur in pneumonia with no distinct signs of dilatation of right ventricle, p. (224).

AT THE VENOUS VALVES.

Venous valve murmur, p. 219, 590.

Produced by incompetence of the venous valves.

Rough quality of the murmur.

Occurrence of a thrill.

Point of maximal intensity over site of origin.

Differential diagnosis easy.

Venous valvular murmur in jugular vein, p. (291).

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH PHYSICAL SIGNS RELATING TO THE LEFT VENTRICLE, p. 46

Early diagnosis of failure of left ventricle is not so easy as that of the right owing to its anatomical situation deep in the chest, pp. 2, 47.

The signs of left ventricular failure are the well-recognised ones of dilatation and mitral systolic murmur.

The lessened cardiac distensibility characteristic of adult life is shown by the increased frequency with which signs of dilatation of the left ventricle occur in overstrain, as compared with early life. Also by the occurrence (with or without left ventricular dilatation) of the ordinary type of dilatation of the right ventricle towards the right with signs of tricuspid regurgitation, pp. 11, 47, 309.

The tendency for the right ventricle to dilate before the left (and especially if upward dilatation of the right ventricle also be present) shows the possession still of some measure of adolescent distensibility, p. 33.

On the other hand, a tendency of heart failure to be accompanied by less dilatation of the right ventricle than might reasonably be expected (considering the degree of overstrain of the left) shows the onset of the relative rigidity which is characteristic of later life. The occurrence of less dilatation of either ventricle than might reasonably be expected shows a further degree of this same relative rigidity, unless there be evidences of myocardial weakness to account for the relative absence of dilatation, p. 147.

In the same manner a complete absence of dilatation of the left ventricle, in spite of overstrain, in a heart strong enough to make dilatation reasonably probable, is an evidence of a considerable degree of the rigidity of later life, p. 86.

The mitral systolic murmur a sign of dilatation of the left ventricle, p. 47.

Its diagnosis from a tricuspid systolic murmur, p. 48.

Its diagnosis from cardio-respiratory murmurs, p. 49, 446.

Sometimes absent in spite of great dilatation, pp. (277), (281), (320).

HEART FAILURE IN VALVULAR DISEASE

Dilatation and hypertrophy necessary for compensation in valvular disease, pp. 47, 389.

Importance of distensibility for the development of compensation, pp. 11, 452.

Serious results of valvular disease in later life or in adherent pericardium where the ventricle is too rigid to dilate, pp. 150, (433), (454), (455), (456).

Estimation of degree of rigidity of the heart wall in valvular disease, p. 150.

Liability of mistaking pallor due to faulty compensation for simple anæmia, p. 453.

See also ‘ Compensation for Valvular Disease ’ (p. 594) and ‘ Mid-diastolic Murmur ’ and ‘ Third Sound of Heart ’ in subject index.

Mitral Systolic Murmur due to organic valvular disease.

Conduction of mitral systolic murmur down the spine, p. 442.

Relationship of left auricle to vertebræ, p. 443.

Estimation of amount of regurgitation by audibility of the murmur down the spine, pp. 444, 447.

Prognostic value of the degree of audibility of the mitral systolic murmur over the lumbar vertebræ, p. 447.

Evidences of the onset of muscular failure in hearts enlarged by valvular disease, p. 110.

Amongst the more evident signs may be noted :—

Defective compensatory dilatation of the heart where there is no reason to suspect rigidity of the heart wall is an indication of myocardial weakness, pp. (65), 150.

Diminution in the size of a heart enlarged by compensatory dilatation and hypertrophy is a sign of deterioration of the heart muscle, pp. 62, 110, (316).

Lessened cardiac dilatation in aortic regurgitation an evidence of myocardial weakness, p. (112).

A lessening in the size of a liver enlarged by chronic venous congestion may be a sign of myocardial failure as well as of improvement of the heart, pp. 111, 112.

A sudden diminution in size of an enlarged liver may be a sign of sudden heart weakness in valvular disease, pp. (63) and (201).

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH HEART FAILURE WITHOUT ENLARGEMENT, p. 50

The force producing dilatation must be the force developed by the ventricular contraction, p. 6.

When the resisting power of the heart walls is greater than the intraventricular pressure which the ventricle can develop, no dilatation is possible; hence in these cases heart failure without enlargement must result, pp. 5, **50, 86, 91.**

The inefficiency of the intraventricular pressure may be due to:—

Abnormally resistant heart walls (see 'Heart Failure without Enlargement in Moderately Strong Hearts'), pp. 86, (87), (89).

Abnormally weak cardiac muscle, pp. 51, 54.

Often both factors are at work, and the part played by each is not always easily determined, pp. 79, **81,** 91, 96, 147.

When there is an absolute inefficiency in the ventricular force—owing to muscular weakness—the amount of blood expelled at each beat (in the face of a normal or an increased arterial resistance) must be lessened proportionally to the lessening in the heart's expulsive power, pp. **51,** 55.

In spite of this lessened output by the ventricle, overdistension of the veins does not usually occur. The veins are, on the other hand, abnormally empty as a rule in myocardial weakness; consequently, when myocardial weakness is well marked there must be a compensatory lessening of the volume of blood in active circulation, proportional to the deficiency in the amount expelled by the ventricles (see 'Blood Volume, Compensatory Lessening of' p. 577), pp. 53, 132.

Where the heart wall is sufficiently soft and yielding, this lessened intraventricular pressure and lessened output is accompanied by a diminution in the size of the heart—as observed clinically—which is proportional to the degree of muscular weakness present.

A group of cases must, therefore, be recognised in which there is—

HEART FAILURE WITH SMALL HEART, p. 54

Compensatory diminution in the size of the heart results in another compensatory advantage—namely, a lessening in the sectional area of the ventricle at the onset of its contraction, whereby, on the principle of the hydraulic press, the discharge of its contents into the aorta is facilitated (the sectional area of the aorta orifice is not likely to be correspondingly lessened), p. 52.

Mechanism bringing about compensatory diminution in the size of the heart, p. 52.

The fullness of a heart depends upon its power of filling itself by the aspiratory force of its expansion.

The force of its expansion is directly dependent on its muscular power both as regards muscular expansion and elastic recoil, p. 53.

In the case of muscular expansion, heart muscles which are too weak to develop adequate contractile force must be still more inadequate as regards expansile force, p. 52.

In the case of mechanical elastic recoil, the vigour of the recoil must be dependent upon the vigour of the contraction causing the recoil and must be somewhat less vigorous when the heart muscle is weak, p: 53.

Therefore, in a heart which fills itself mainly by its *muscular* aspiration—when the heart muscle is weak—its power of filling itself will be at least as defective as its power of emptying itself, p. 53.

IN ADOLESCENCE.

Therefore, in early life when (owing to the immaturity of the fibrous tissues) the heart does depend almost entirely upon its muscular aspiration (p. 183), incomplete filling may be expected to be proportional to the inefficacy of its expulsive power, p. 53.

Therefore, well-marked and adequate compensatory diminution in the size of the heart will accompany myocardial weakness, p. 56.

Clinically, in adolescence, marked diminution in the size of the heart *is* found in cases of myocardial weakness. The smallness of the heart appears to be proportional to the amount of weakness present, pp. (65), (68).

IN ADULT AND LATER LIFE.

In hearts possessing elastic recoil in addition to muscular expansion, less extreme interference with the filling of the heart will occur, and, therefore, compensatory diminution in the size of the heart will be less complete in adult and later life than in adolescence, pp. 54, 73, 80.

The greater the amount of elastic recoil possessed by the heart the less perfect will the compensatory incompleteness of the filling of the heart and the less its reduction in size, p. 80.

As the possession of elastic recoil depends upon the rigidity or otherwise of the fibrous elements of its wall (i.e. its distensibility), it can be asserted that the distensibility of a heart that is muscularly weak may be estimated by the amount of diminution in the size of the heart that occurs for any given degree of myocardial weakness. The distensible heart will show much diminution ; the relatively rigid one but little, p. 81.

Compensatory diminution in the size of the heart is interfered with in another manner by the maturity of the fibrous tissues upon which the possession of elastic recoil depends. This same maturity (or relative rigidity) of the tissues of the heart wall tends to make it retain its normal size and shape, instead of remaining only partly filled, as in the case where its tissues are softer, p. 79.

In later life, where a considerable degree of this rigidity may occur, there may be no lessening at all of the size of the heart in myocardial weakness ; just as under similar circumstances heart failure in a strong heart may occur without any dilatation, p. 80.

It may, therefore, be asserted that the distensibility of the heart in adolescence and early life is shown by the degree to which the moderately strong heart dilates in the face of overstrain and by the degree in which the weak heart diminishes in size on the occurrence of myocardial weakness, p. 81.

The degree of rigidity of the heart in adult and later life is shown by the extent to which a strong heart fails to dilate under circumstances that might be reasonably expected to produce dilatation, pp. 86, (87).

Or, in the case of a weak heart, by the extent of its failure to diminish in size in the face of well-marked myocardial weakness, p. 81.

Certain consequences follow this imperfect compensatory diminution in size.

The compensatory diminution in the size of the ventricle being less complete than in a softer heart there will be incomplete emptying of the ventricle with each systole, p. 82.

The possession of elastic as well as muscular aspiration will render the ventricle liable to draw into itself more blood than it is able to expel, p. 53.

In adolescence and early life, anginal pain, from ventricular over-distension, is extremely rare—apart from valvular disease—because the heart cannot overfill itself. If too weak to empty itself properly it is also too weak to fill itself, p. 54.

In adult and later life increasing rigidity of the cardiac tissues increases the liability to overfilling and to the occurrence of anginal pain, pp. 82, 148.

Also as regards sudden death from asystole.

For the above reason this is very rare in the myocardial weakness of early life ; but in later life the liability to sudden death in myocardial disease increases as age advances, p. 54.

HEART FAILURE WITHOUT ENLARGEMENT IN ADOLESCENCE

CLINICAL FEATURES of heart failure without enlargement in distensible hearts—namely, in heart failure with small heart.

As has already been pointed out, where the heart is distensible (as it is in adolescence) severe muscular failure is accompanied clinically by a diminution in the size of the heart.

GENERAL SYMPTOMS.

Extreme weakness. Faintness on the slightest exertion – sometimes even on raising the arm from the side.

Pulse very weak and small, pp. 35, 56, 59, 95.

Special character of pulse in myocardial weakness.

A high tension pulse of very low pressure and very small volume, p. 59.

Compensatory contraction of the peripheral arterioles in extreme heart weakness, p. 59.

Illustration: the pulse characteristic of severe enteric fever, p. 59.

Very weak or absent impulse in jugular bulb, pp. 97, (98).

Emptiness of the neck veins, p. 61.

In the distensible heart of adolescence, full reliance can be placed on the diminution in the size of the heart itself and in the evidences of compensatory diminution in the blood volume given by the rise of the diaphragm and diminution in the size of the absolute liver dullness, p. 56.

PHYSICAL SIGNS relating to the heart itself. Cardiac impulse—weak or absent, pp. 56, 96.

Evidence of diminution in the size of the heart. Cardiac dullness small or absent, pp. 56, 60, 96, 187.

Diminution not due to increase in size of lungs, as is shown by diminution in their size, pp. 57, (98).

Complete disappearance of dullness, when it occurs, is probably partly due to falling backwards of the small and badly filled heart, p. 56.

Cases showing extreme smallness or absence of cardiac dullness, p. (65), (68), and (118).

A lessening in the size of a dilated heart may be the sign of the onset of myocardial weakness, p. (316).

Feebleness of heart sounds, pp. 60, 96.

Loudness of pulmonary second sound of importance, p. 96.

Evidences of a COMPENSATORY DIMINUTION in the VOLUME OF BLOOD in circulation, p. 55, 577.

Diminution in the volume of the thoracic contents from underfilling of the lungs and great vessels with blood, pp. 57, 163.

Diaphragm raised, pp. 57, 163, 180. Shown by high gastric resonance, pp. 57, 164.

Upper level of liver dullness high, pp. 57, 165.

Signs that the liver is underfilled with blood, pp. 187, 191.

Diminution in its area of absolute dullness, pp. 95, &c.

Diagnostic value of high diaphragm and small liver dullness in heart weakness, pp. 56, 72, (97), (195), (326).

Diminution in the absolute dullness of the liver due to causes other than heart weakness, p. 152, **202**.

Disappearance of these signs as the heart regains its muscular strength, pp. (196), &c.

HEART FAILURE WITHOUT ENLARGEMENT IN ADULT LIFE

CLINICAL FEATURES of heart failure, without enlargement, in the moderately distensible heart of adult life.

In adult life the lessened distensibility of the heart lessens the amount of its diminution in size when very weak, and also lessens the extreme movements of the diaphragm, which are noticeable with the softer fibrous tissues of early life, pp. 72, 79.

Symptoms of general weakness, smallness of pulse and feebleness of heart's impulse and heart sounds are of value as in adolescence, p. 91.

With the lessened possibility of change in the size of the heart, more dependence is placed upon the available signs of compensatory diminution in the blood volume, pp. 51, 61, 94.

Of these the most reliable are :—

Rise in the level of the gastric resonance.

Evidences of lessened volume of the thoracic contents given by hollowing of the supra-clavicular fossæ (where the diaphragm is too rigid to rise adequately), pp. 94, 134, 187.

Evidences of weak and ineffective action of the right ventricle given by feebleness and smallness or even absence of the jugular bulb pulsation, pp. (98), 133.

Emptiness of the veins of the neck may be of value—especially in cases where fullness might be expected, pp. 98, (133).

Diminution in the size of the absolute liver dullness. The greater the distensibility the more nearly will the clinical features approximate to those of adolescence. This is also true of heart failure in later life, p. 195.

In a moderately distensible heart, failure due to myocardial weakness can often be diagnosed by the *absence* of the dilatation and venous fullness or of right ventricular overaction, under circumstances calculated to produce signs of right ventricular overstrain—such, for instance, as pneumonia, pp. (97), (105), or in valvular disease, pp. 62, 64.

Assistance may be given in the diagnosis of myocardial weakness by the presence of pain in the calves on exertion from faulty blood supply to the muscles, pp. 92, 467, (470).

Also coldness on exertion from faulty blood supply, pp. (365), 472, (473), (475).

ENLARGEMENT OF A SMALL HEART A SIGN OF RETURNING STRENGTH

From what has just been said it follows that :—

Increase in the size of a heart that has diminished in size, owing to myocardial weakness, will be a sign of returning strength, or in a weak heart, dilatation may be a sign of returning strength, p. 63.

Illustrative cases, pp. (64), (294), (304).

Similarly, the return of cardiac strength may be measured by the increase in the size of the absolute liver dullness, p. 64.

Illustrative cases, pp. (66), (195), &c.

Also by the return of the diaphragm (as shown by the level of the gastric resonance) towards its normal position, pp. (118), (196), &c.

Return of strength will also be shown by lessening of the upward dilatation due to recumbency, p. 315.

Also, on the gradual return of strength in a distensible heart, a point is usually reached when the strength of the stronger part of the right ventricle exceeds that of the weak part of the anterior wall ; and even though the patient be confined to bed, upward dilatation with abnormal pulsation occurs, fig. 46, p. 197.

Upward dilatation of the right ventricle a sign of returning strength after myocardial weakness.

Illustrative cases, pp. (102), (294), (304), &c.

Thus the physical signs of the return of strength in a weak heart are the same as those of heart failure in a strong heart, pp. 63, 71.

For other cases, see pp. (64), (100), (157), (182), (196), (316).

A Summary and Index of Arguments and Facts brought forward in Connection with Early Diagnosis of Heart Failure in Later Life

The early diagnosis of heart failure as age advances becomes more difficult than is the case earlier in life, for the following reasons :—

I. Owing to the increasing rigidity of the cardiac and pericardial tissues in later life, changes in the size of the heart, indicative of heart failure, take place less readily and in extreme cases not at all, pp. 72, 79.

(*a*) Dilatation, when it does occur, will be less in amount for any definite degree of overstrain, p. 87.

(*b*) There will be a larger proportion of cases where the diagnosis will be rendered more difficult by the fact that cardiac failure is not accompanied by any clinical evidences of dilatation, pp. 79, 87.
Early diagnosis of failure in such cases, p. 90.

(*c*) There will be a lessened likelihood, in the case of myocardial weakness, that extreme heart failure will show itself by diminution in the size of the heart, p. 80.

Estimation of the degree of rigidity possessed by the heart wall is of great importance in later life, but of considerable difficulty, p. 147.

In the absence of signs due to changes in the size of the heart, increased dependence has to be placed upon the less certain signs given by changes in the volume of blood in circulation, and especially upon the evidences of fullness or emptiness of the auriculo-venous reservoir and of the lungs.

II. The increasing rigidity of the fibrous tissues of the body generally will interfere—

(1) With the physical signs whereby overfullness of the auriculo-venous reservoir, and of the lungs, manifests itself in cases of cardiac failure with a moderately strong heart; and also—

(2) With the physical signs, whereby compensatory diminution of the blood volume manifests itself in cases of very considerable cardiac weakness, p. 72.

(*a*) Alterations in the level of the diaphragm will take place less readily, p. 72.

(*b*) Fullness or emptiness of the supra-clavicular fossæ, as evidence of increased or diminished volume of the intrathoracic contents, while not being so greatly interfered with as is the case with changes in the average level of the diaphragm, will, nevertheless, be lessened by increasing rigidity of the fibrous tissues.

(*c*) Changes in the size and flaccidity of the liver will be less marked, and less extreme variations in the size of its absolute dullness may be expected.

III. There are difficulties in appraising the true value of the radial pulse and of the blood pressure as estimated by the sphygmomanometer, owing to the frequency of abnormalities of the blood pressure in later life, p. 143.

Difficulties consequently occur in estimating what may be called the internal work that the heart is doing, p. 73.

There is need for very carefully estimating the bearing of such high arterial resistance, as may be present, upon the heart's action, p. 74.

1. Increased arterial resistance may be pathological, due to arterial disease or spasm, and the rise in blood pressure so caused may be throwing an extra burden on the heart, p. 143.

(*a*) Owing, however, to abnormal rigidity of the heart wall no evidence of this fact may be given by cardiac dilatation.

(*b*) Owing to myocardial weakness no evidence may be given by increased vigour of cardiac action of the extra burden that is being thrown upon the heart.

2. Increased arterial resistance may be compensatory in nature, because the heart is better nourished by a high than by a low blood pressure (owing to blocking of the coronary arteries or other cause), p. 144.

3. High blood pressure, due to increased arterial resistance, may be (owing to the presence of myocardial weakness) less than is necessary for adequate nourishment of the heart. In such a case as this a rise in the already high blood pressure accompanies the return of the heart to a more normal mode of working, p. 146.

Complexity of the inter-relationship in later life between arterial resistance—the amount of work done by the heart and the distensibility of the heart wall, p. 142.

The radial pulse. Its diagnostic value, p. 143.

Sphygmo-oscillometer. Its great value as showing the type of high blood-pressure present in any case, p. 145.

Compensatory and non-compensatory high blood pressure show a different type of oscillation, p. 145.

In heart failure with high blood pressure, a further rise may sometimes be followed by improvement in the heart, p. 146.

Abnormality of the blood pressure may be due to a lessening of the elasticity of the first part of the aorta, p. (109).

IV. Difficulties associated with myocardial weakness due to interference with the blood supply by arterial disease affecting the coronary arteries, p. 75.

The myocardial weakness of later life is much more difficult to recognise than that of adolescence and early adult life.

1. The rigidity of the cardiac tissues in later life lessens the likelihood of myocardial weakness being accompanied by recognisible reduction in the size of the heart.

2. The increased fibrous strength of the heart wall which is apt to characterise later life is very apt to be mistaken for muscular strength, pp. 34, 150.

A heart which does not dilate when subject to overstrain is not infrequently (in later life) weaker muscularly than a heart which does dilate.

Need for distinguishing clinically between the fibrous strength of the heart wall and its muscular power, and deciding in a case of heart failure without enlargement whether its non-dilatation is due to rigid walls or weak muscle, p. 73.

3. Difficulty in estimating the amount of power developed by the heart muscle in later life (easy in early life), p. 142.

4. Myocardial weakness due to disease of the coronary arteries is apt to be much more insidious in its onset than that of adolescence and early adult life, which is usually infective in character and has some obvious cause, p. 153.

5. Cardiac weakness of this type is apt to increase so gradually that compensatory changes in the circulation and the patient's habits may prevent the appearance of any obvious symptoms in its earlier stages, p. 75.

6. Disease of the coronary arteries often interferes with the nutrition of only a portion of the heart muscle, and not the whole as in infective myocardial weakness, p. 75.

Difficulties arise when one ventricle is strong and the other weak, p. 153.

Need for distinguishing between the phenomena due to weakness of the right ventricle and those due to the left, p. 154.

Dyspnœa a symptom of right ventricle weakness, pp. (154), (156).

Faintness and weariness a sign of weak left ventricle, pp. 121, (124), 154.

Leg pains on exertion a sign of weak left ventricle, pp. 92, 467, **469**.

Coldness on exertion a sign of myocardial weakness of left ventricle, p. (365), 471.

Signs when left ventricle strong and right one weak, pp. (109), (154), (156).

Signs when right ventricle strong and left one weak, p. (107).

Strong collapsing radial pulse in severe fatty degeneration of right ventricle with strong left ventricle, p. 154.

7. Difficulties associated with the diagnosis of the onset of myocardial weakness in general, or with the return of cardiac vigour on its subsidence.

The physical signs of failure in a muscularly weak heart are the reverse of those in a stronger one, p. 71.

In a moderately distensible heart in adult and later life dangerous heart weakness may be shown by the *absence* of the ordinary signs of heart failure, p. 71.

Cases of pneumonia illustrating this, pp. (97), (106).

In a moderately strong heart which is showing signs of failure with enlargement and venous plethora, the signs of the onset of myocardial weakness are, in many respects, the same as those of returning strength, p. 110.

Illustrative case. Lessened right ventricular pulsation: at one time showing improvement, at another dangerous weakness in the same patient, pp. (106), (141).

Diminution in the size of a liver enlarged by tricuspid regurgitation, a sign of deterioration and not improvement, pp. (63), (110).

The signs of the return of strength after myocardial weakness in a weak heart may be the same as those of the onset of heart failure in a moderately strong heart.

Summary of phenomena, which may indicate either increasing or decreasing weakness of the heart, p. **160**.

V. Difficulties arising from the fact that greater dependence has to be placed upon GENERAL SYMPTOMS of heart failure, and the fact that they are often misleading.

Breathlessness, pp. **92**, **113**.
  Tachycardia in its relation to dyspnœa, p. **113**.
  Importance of breathlessness as showing a muscularly weak right ventricle, pp. **93**, (155), (156).

Palpitation, p. **114**.
  Functional palpitation and irregularity simulating organic disease, p. (115).
  Reflex palpitation simulating cardiac overstrain, p. (115).
  Colon dyspepsia a cause of palpitation, p. (116).
  Nocturnal attacks of palpitation suggest a reflex colon origin, p. (116).
  Palpitation as a cardiac habit after recovery from severe asthenia, p. (117).
  Habit palpitation after influenza and after diphtheritic cardiac weakness, p. (119).

Faintness, pp. **92**, (121).
  Fainting and asthenia of labyrinthine origin liable to be mistaken for a sign of dangerous heart weakness, pp. **122**, **123**.
  Faintness of cardiac origin diagnosed as of gastric origin, p. (124).
  Danger of fatal syncope in cardiac asthenia from micturition in erect attitude, p. **126**.
  Sudden death when right ventricle is weak and left one strong—probably due to fainting from deficient supply of blood to the left ventricle, p. **125**.
  Sudden death when right ventricle stronger than the left apt to be due to asystole from an overfilling of the weak left ventricle.

Anginal pain, p. 126.

Liability of mistaking false angina for true.

Vasomotor angina of reflex origin in colon dyspepsia, pp. (127), (130).

Value of nitroglycerine in reflex angina, pp. (127), (130).

Anginal pain produced by palpation of an irritable colon, p. 129.

Nocturnal occurrence of attacks suggests colon origin, p. 131.

Anginal pain not likely to occur in early life because of compensatory underfilling of the ventricle, p. 53.

Anginal pain more likely to occur the greater the relative rigidity of the heart, because the presence of mechanical elastic recoil interferes with the compensatory underfilling of the heart, pp. 82, 148.

Leg pains and intermittent claudication a sign of myocardial weakness, pp. 92, 469.

SUMMARY OF EVIDENCES OF HEART FAILURE IN LATER LIFE

Clinically, in the diagnosis of heart failure with weak heart in later life, reliance must be placed upon the symptom of breathlessness in the first place.

Faintness or vertigo, if of cardiac origin, is of serious import, p. 121.

A very weak or low pressure pulse is important evidence. If normal or strong its meaning must be carefully analysed, p. 143.

Weak or absent jugular bulb pulsation is of great clinical value. A strong and full jugular pulse without any cardiac dilatation suggests undue cardiac rigidity, p. 134.

The venous phenomena are of much importance (see p. **132**, and table, p. 137).

Amongst physical signs, most dependence must be placed upon those of a compensatory diminution in the volume of blood in circulation—especially

(1) High gastric resonance, 56, **163, 185**.

(2) Hollowness of supra-clavicular fossæ and emptiness of the neck veins, p. 134.

(3) Diminution of the absolute liver dullness (when

present) suggesting altered conductivity to percussion vibrations by lessened tension of its tissue from underfilling with blood, p. 191.

4. Weak heart sounds and impulse are important, if not explicable by changes in lung or thoracic wall, p. 152.

As a result of what has been said as to the occurrence of heart failure without enlargement in a strong but relatively rigid heart, and also in a weak and rigid heart, it follows that it is possible to have a case of heart failure where the size and mode of action of the heart appear normal in every respect, p. 148.

Such a possible case analysed, p. 149.

Difficulties in diagnoses in later life. Summary of opposing and apparently contradictory statements, p. 160.

A SUMMARY AND INDEX OF ARGUMENTS AND FACTS BROUGHT FORWARD IN CONNECTION WITH COMPENSATORY DIMINUTION OF THE BLOOD VOLUME

In cases where the amount of food is limited, as in the treatment of gastric ulcer, or where the absorption of nourishment is interfered with, as in enteric fever, a progressive diminution in the volume of the thoracic contents is noticeable, as evidenced by a rise in the diaphragm, pp. 168, 178.

The only variable substance in the thorax is the blood contained in the heart and the blood-vessels—especially those of the lungs, p. 179.

Therefore a diminution in the bulk of the thoracic contents must mean a diminution in the amount of *blood* contained in it.

Further, on post-mortem examination of, for instance, a case of enteric fever dying of asthenia from non-absorption of food, the diaphragm is found to be abnormally high and 'the organs of the body extraordinarily dry and bloodless,' p. 168.

These and similar observations point to the fact that where there are insufficient materials available for the formation of the normal amount of blood of the proper specific gravity, an abnormally small amount of blood of a good specific gravity is maintained in circulation rather than a nearly normal amount of an abnormally low specific gravity, p. 180.

In other words: there is clinical evidence that the total volume of the blood in circulation is reduced when the essential constituents of the blood fall below a certain definite minimum.

In the case of the specific gravity, observation would probably determine what the minimum was, below which the specific gravity of the blood was not allowed to fall, p. 180.

This is a fruitful field for research.

This compensatory diminution in the volume of the blood can be observed clinically under the following conditions:—

1. Limitation of ordinary food, as in the treatment of gastric ulcer, pp. **169,** 194, **204**.
   Starvation due to disease, e.g. enteric fever, p. 168.
   Malnutrition from any cause, e.g. anæmia, pp. 176, 203.
2. Limitation of water.
   In cases of diarrhœa and vomiting, p. 203.
3. Loss of blood from severe hæmorrhage, pp. 175, 203.
4. Deficiency of thyroid secretion, pp. 178, 195, **206**.
5. In cases of neurasthenia from the deficiency of some unknown material necessary for the nutrition of the nervous system, pp. 177, 195, 207.

These observations seem to show that there are certain essential constituents of the blood and that *each* has a minimal percentage below which it is not allowed to fall. In the case of a great deficiency, the total volume of blood must be lessened in order that an adequate percentage composition may be maintained in spite of the inadequate supply of the particular substance.

COMPENSATORY DIMINUTION IN THE BLOOD VOLUME DUE TO CARDIAC INEFFICIENCY

Not only is the total volume of the blood diminished when materials for its formation are deficient, but the amount of blood in circulation is compensatorily diminished when the strength of the heart is insufficient for the circulation of the normal amount. Observation of cases of cardiac weakness gives precisely the same signs of deficiency in the blood volume as those just described, pp. 53, 180, 185.

In cardiac weakness, the compensatory emptiness of the blood-vessels does not necessarily imply deficiency of the *total* amount in the body.

There are evidently reserves which can be drawn upon if desired. Case proving this, pp. (141), 186.

The rationale of compensatory diminution in the amount of the circulating blood in heart weakness is clear. The work the heart has to do is cut down proportionally to its power of doing it. The heart's load is made proportional to its strength, pp. **50**, 55, 180, 186.

The mechanism whereby this is brought about is simple. In the distensible heart of adolescence (which depends on its muscular expansion alone for its power of filling itself) the same weakness which interferes with the emptying of the heart will interfere equally with its power of filling itself, p. 52.

In the less distensible heart, which possesses mechanical elastic recoil, although the compensatory diminution in the filling may be less complete, it nevertheless takes place, for the amount of elastic recoil is directly dependent on the vigour of the preceding contraction, pp. 53, 79.

Evidences of a diminution in the volume of the blood are of the utmost value in the diagnosis of myocardial weakness, and especially so in adults and in later life where, owing to increasing rigidity of the heart, less dependence than in early life can be placed upon the condition of the heart itself, p. 186.

The main CLINICAL SIGNS of compensatory diminution of the blood volume depend upon the lessened distension with blood of the more elastic and vascular of the viscera which are readily studied clinically.

Of these the lungs, the heart, and the liver are of most importance, p. 187.

1. Diminished distension of the lungs with blood (in conjunction with a similar condition of the heart and great

veins) causes what is, in effect, a negative pressure within the thorax, and this shows itself clinically by changes in the less rigid portions of its walls—that is by—

(1) A rise in the level of its floor—namely, the diaphragm, pp. 57, **180**; and—

(2) By a sinking-in of the soft tissues which fill in the space which is enclosed by the first rib on either side—namely, the tissues which occupy the supra-clavicular fossæ, pp. 94, 134, 139.

(1) A rise in the level of the diaphragm.

This may be measured clinically by noting—

(*a*) The level of the gastric resonance (normally, the sixth interspace) as marking its level on the left side, pp. 57, **164**, (171), &c.

(*b*) The upper level of the liver dullness as marking its level on the right side, p. 165.

(*c*) The lower level of the liver dullness may also rise owing to the organ being drawn up under the highly arched diaphragm. This change is less certain and of less diagnostic value than the two above mentioned and need not be further dealt with.

In the first two or three decades of life this alteration in the diaphragm takes place readily owing to the softness of the fibrous tissues. It constitutes a sufficient guide to the amount of blood in circulation in the thoracic viscera.

In heart weakness the level of the diaphragm must be carefully watched from day to day. It is of much diagnostic value. The gastric resonance sometimes reaches up to the fourth rib, pp. (68), (118), and (182).

The changes in level of the liver dullness are apt to be interfered with by the inertia of the liver; but, nevertheless, in young subjects with extreme heart weakness the upper border of its relative dullness may rise as high as the third rib, p. (182).

In adult and later life, changes in the level of the diaphragm are less extreme, owing to the increasing rigidity of the fibrous tissues, pp. 72, 94.

Changes in the upper level of the liver dullness are in later life too slight to be of clinical value owing to this cause; but changes in the level of the gastric resonance are, as a rule, still of diagnostic importance, p. 94.

(2) Recession of the tissues in the supra-clavicular fossæ as a sign of diminished circulation in the thoracic viscera.

While the fibrous tissues still remain distensible, the changes in the diaphragm are adequate to compensate for any degree of diminution in the bulk of the thoracic contents, therefore no hollowing of the supra-clavicular fossæ is noticeable.

In later life, however, the greater rigidity of the diaphragm makes it less able to compensate by its mobility for alterations in the size of the thoracic viscera. Under these circumstances sufficient negative pressure may be developed by compensatory diminution of the blood volume to cause hollowing of the supra-clavicular fossæ. Hollowing of the supra-clavicular fossæ a valuable sign of heart failure in later life, pp. **94, 134, 139.**

2. Diminution in the size of the heart a sign of compensatory diminution in the blood volume, p. **188.**

This diminution in the size of the heart as observed clinically is of far greater diagnostic importance where the lessened blood volume is due to cardiac weakness than when due to lessened volume from nutritional and allied causes. It is sometimes extreme in great myocardial weakness, causing, even, complete disappearance of both absolute and relative cardiac dullness, pp. **54,** (68), and (118).

This is a sign that is most evident when the cardiac tissues are soft in the first two or three decades of life.

In adult life the increasing relative rigidity of the cardiac tissues, while not interfering very greatly with compensatory diminution of the blood volume in cardiac weakness, does interfere with the diminution in the size of the heart, pp. **81, 82.**

In later life the relative rigidity of the heart may entirely prevent any observable diminution in its size, even though there is considerable compensatory diminution in the volume of the blood which it is able to keep in circulation.

3. Diminished distension of the liver with blood causes certain changes which are observable clinically and are of great diagnostic value in determining the amount of blood in active circulation, p. **185.**

These changes show clinically by a diminution in the area of the liver dullness involving both its relative and absolute dullness.

The changes in absolute dullness are more marked than those in the relative dullness and are of much greater diagnostic value.

Diminution in the area of liver dullness from this cause is brought about—

(1) By an actual diminution in the size of the liver owing to its lessened distension with blood, pp. 188, 189.

(2) The insufficient distension causes the liver to be more flaccid than normal, and its thin edge is apt to fall back and allow the approach of the resonant intestines to the ribs. Case tending to prove the flaccidity of the liver in a case of voluntary starvation for gastric ulcer, pp. 190, (205).

(3) Neither of these explanations is adequate to explain the more extreme cases of absence of absolute liver dullness which are met with clinically, p. 191.

It is certain that *over-distension* of the liver tissue with blood, in chronic venous congestion, deprives the liver tissue of its power of masking the resonance of the underlying intestines and thus increases its conductivity to percussion vibrations, p. 24.

It seems probable that the *lessened* tension of its tissue which results from *underfilling* with blood also has this power of increasing its power of conducting percussion vibrations to and from the underlying resonant intestines.

Clinically, the absolute dullness of the liver varies in extent in such a way as to show that the degree of its diminution is an absolutely reliable guide to the degree to which the liver tissue is relaxed from underfilling with blood, p. (195).

These changes which indicate the degree to which the blood volume is being compensatorily diminished are of great clinical value in estimating the effect of starvation or rectal feeding on a patient. Thus showing the safety or otherwise of its continuance, p. 205.

But they are of much greater clinical value as showing the degree in which the volume of blood in circulation in the liver is compensatorily lessened when there is muscular weakness of the heart, p. 185.

In cases of increasing cardiac weakness, the diminution of the absolute liver dullness is noticeable from day to day as the heart gets weaker, and in the same way its return towards the normal size is an accurate measure of the return of the heart's strength, pp. (195) to (199).

Although this does not always take place, the size of the absolute liver dullness does as a rule give very valuable indications as to the amount of blood the heart is able to keep in circulation.

The regular way in which variations in the area of absolute liver dullness respond to variations in the heart's activity suggests that this phenomenon of lessened liver dullness is mainly due to an alteration in the degree of distension of the liver tissue rather than to the other factors mentioned, p. 191.

Cardiac asthenia of rheumatic origin and small liver dullness, p. (195).

4. Evidence as to the emptiness of the auriculo-venous reservoir must be looked for by examination of the one place where direct clinical study of its condition is possible—namely, the so-called jugular bulb, where the external jugular vein opens into the subclavian, pp. 133, 137.

A lessening in the volume of the jugular bulb pulsation (as palpable behind the clavicle) is suggestive of compensatory diminution in the fullness of the reservoir.

Where lessened vigour of pulsation accompanies the lessening in volume it suggests that cardiac weakness is the cause of the compensatory lessening of the blood volume, p. 97.

Where cardiac weakness is considerable the jugular bulb pulsation is no longer palpable, pp. 137 and 138.

5. Emptiness of the veins is also a sign that there is compensatory diminution in the volume of blood in circulation, p. 132.

In the case of the veins at the root of the neck, important evidence may be given by their emptiness in the case of cardiac weakness, pp. 57, 61, 95, 187.

Emptiness of the neck veins is of considerable diagnostic importance when it occurs under circumstances that might be expected to result in their over-distension.

Emptiness of the neck veins in pneumonia a sign of dangerous myocardial weakness, pp. (97), (105).

Emptiness of the venous system shown by the occurrence of dyspnœa on the application of the armlet of the sphygmomanometer, p. 140.

SUMMARY OF FACTS AND ARGUMENTS IN CONNECTION WITH THE AURICULO-VENOUS RESERVOIR

It is a compensation reservoir for the supply of blood to the right ventricle, p. 132.

ANATOMICAL CONSIDERATIONS

The reservoir is constituted by—

1. The right auricle.
2. The superior vena cava, innominate veins, and the first part of the subclavian veins.
3. The vena azygos and its branches.

It is enclosed within certain valves—namely :—

1. The Eustachian valve guarding the inferior vena cava.
2. The venous valves which prevent a reflux into the distal veins. Chief ones are those in the subclavians and those at the mouth of the internal and external jugular veins, and those of the azygos system.

PHYSIOLOGICAL CONSIDERATIONS

TONIC ACTIVITY of its walls.

1. The muscular walls of the auricle and the veins are possessed of tonic activity whereby the blood in the reservoir is maintained at a definite minimal pressure, pp. 133, 503.
2. In order to do this the muscular walls must respond by increased tonic activity to any tendency to the lowering of the blood pressure. The muscular fibres must therefore be sensitive to mechanical stimulation by relaxation of their tension, p. 507.
3. The muscular walls, in virtue of their tonic activity, also endeavour to counteract the tendency to dilatation

of the reservoir which would result from any excess of blood pressure within it.

4. To accomplish this the muscular walls of the auricle and the veins are sensitive to mechanical stimulation, by stretching, and respond by increased tone to any force tending to dilate it, pp. **37**, **504**, **506**.

Contractile activity of the muscular walls of the reservoir. As regards the auricle.

1. It contracts in response to nodal stimulation at a time when it is only half-full, just prior to the ventricular systole—the so-called auricular systole. The most effective part of this contraction—is the contraction of the auricular appendix whereby (*a*) the filling of the ventricles is completed, (*b*) the auriculo-ventricular valves are made tense in preparation for the ventricular systole, and (*c*) in all probability the stretching of the ventricular walls by the sudden entry of blood from the auricle adds a *mechanical* 'stimulus-to-contract' to the *nodal* one. The contraction of the appendix is the spark that fires the powder and determines the exact time of the occurrence of the ventricular systole. Irregularity of rhythm characterises a fibrillating auricle, p. **529**.
2. The muscular wall of the auricle is sensitive to mechanical stimulation by stretching of its walls, and a true contraction may result if the distension be sudden enough and powerful enough (such a contraction may be detected by the cardiograph in right ventricular tracings if the distending force be not so powerful or so prolonged as to prevent the auricle from throwing blood into the ventricle), pp. **504**, **515**.
3. The auricle wall can also be stimulated mechanically to a prolonged contraction if the distending force be maintained. Thus in tricuspid regurgitation, in the rare cases where tracings can be taken from the body of the auricle, it is found sometimes that the

auricle does not become distended by the regurgitating blood till towards the end of the ventricular systole, pp. 509, 521.

4. It is also probable that the muscular walls of the auricle may be stimulated to develop a true contractile act (as distinct from a mere increase of tonicity) by a sudden *relief of tension* such as takes place when its contents are aspirated into the ventricle during the expansion phase of the cardiac cycle.

As regards the venous portion of the reservoir.

1. The walls of the great veins which constitute a portion of the reservoir are stimulated to contract by a distensile force of adequate suddenness and power. Such a contraction may be *observable* in tracings taken over the jugular bulb, provided that the force is not sufficiently powerful to overcome the muscular power of the veins nor sufficiently prolonged to prevent the contraction from emptying the veins. (Such a contraction may be demonstrable in cases where an extra systole causes forcible tricuspid regurgitation of small volume), p. 506, fig. 160, p. 513, point 10.
2. These veins are also stimulated to a true contractile act by a sudden relief of tension within them. Such a contraction is observable in tracings taken over the jugular bulb when a distended reservoir is suddenly emptied during the auricular expansion phase which follows its systole, p. 508, fig. 160, point 2.

As regards the relationship of the right ventricle during its relaxation phase to the auriculo-venous reservoir.

During its 'relaxation phase' the right ventricle contains the blood aspirated into it during its expansion phase, and this blood does apparently constitute a part of the contents of the reservoir. The walls of the right ventricle are, during the 'relaxation phase,' maintained in a state of tonic contractility and the blood within it is maintained thereby at the same pressure as that in the auriculo-venous reservoir proper, p. 513.

Moreover, there is reason to believe that during its relaxation phase the walls of the ventricle are sensitive to mechanical stimulation and a true contraction can be originated by sudden distension with blood, p. 497.

During the relaxation phase it is probable that the tonic power of the walls of the ventricle is less than that of the auricle and the veins, and that by this means the blood which has been aspirated into the ventricle is maintained within it.

Other physiological points in connection with the auriclo-venous reservoir in health and disease.

1. The venous valves may be considered an integral part of the cardiac mechanism.
    (*a*) Their presence admits of the maintenance of a moderately high blood-pressure within the reservoir.

    The presence of this definite blood pressure within the reservoir is evidenced by the fact that when tracings are taken simultaneously over the reservoir behind the clavicle and over the right ventricle the conduction of impulses through the blood in the reservoir is practically instantaneous, p. 503.

    (*b*) They admit of the utilisation of the respiratory movements for the filling of the right ventricle, p. 13.

    Inspiration tends to fill the reservoir, and expiration to empty it into the ventricle. As evidence of this, the first cardiac beat that occurs during expiration is (so far as the right ventricle is concerned) more effective than those during the remainder of the respiratory cycle; for cardiac sounds and murmurs, and cardiac pulsation due to this beat, are demonstrably louder than the others, p. 13.

    The effect of deep sighing respirations in aiding a failing right ventricle depends upon this phenomenon, p. 13.

(*c*) They, in conjunction with the tonic contractility of the walls of the veins and of the auricle, hinder tricuspid regurgitation and thus can give support to the right ventricle when overtaxed, p. 37.

2. The tonic contractility of the auricle and veins retains in the relaxed ventricle the blood that it aspirated into itself during its expansion phase.
3. The fact that the superior vena cava is in close anatomical contact with the ascending aorta, results in the compression of the vein by the sudden distension of the first part of the aorta which takes place at the commencement of the ventricular systole.

   The sudden rise in venous pressure due to this compression is evidenced in tracings taken over the so-called jugular bulb by a systolic wave in the venous tracing which is synchronous with the systolic wave in the ventricular tracing due to the return of blood from the distended first part of the aorta (i.e. with the 'first aortic' or recoil wave), pp. 512, 531.
4. The functions of the azygos vein in relation to the auriculo-venous reservoir.

   In consequence of the ineffectiveness of the valves in this vein and the relationship of its intercostal tributaries with the anterior thoracic veins, it is probable that it acts as a safety-valve-like outlet to the reservoir.

Physiology of the auriculo-venous reservoir in relation to the phases of the cardiac cycle.

These are fully summarised at p. 528.

Pathological changes in the reservoir.

1. As regards the right auricle.

Dilatation in tricuspid incompetence.

Dilatation takes place to the right and backwards, and, owing to its association with dilatation of the ventricle, a dilated auricle very rarely indeed comes into close relationship to the anterior chest wall, p. 41.

Its dilatation may lead to incompetence of the

Eustachian valve and engorgement of the hepatic vein, p. 39.

Dilatation sometimes very excessive, p. (210).

The membranous lower portion is sometimes greatly dilated, p. (210).

2. As regards the great veins.

Their dilatation in tricuspid regurgitation sometimes give rise to distinct pulsation in the episternal and supra-clavicular fossæ; also in the veins of the neck through incompetence of the valves.

The dilatation of the superior cava is less at the point where it passes through the pericardium than elsewhere, and thus a constriction is caused in the vein, at which a murmur can arise. This may be called the 'superior vena cava murmur,' pp. 44, 218.

CLINICAL PHENOMENA ASSOCIATED WITH PATHOLOGICAL CONDITIONS OF

I. The right auricle.

(1) When dilated it is very rarely indeed recognisable by palpation or percussion anteriorly, owing to the direction in which it usually dilates, p. 41.

When considerably dilated there may be increased loudness of the heart sounds over the ninth and tenth interspaces to the right of the spine or in the right axilla, p. 42.

(2) Dilatation of the lower part of the auricle may lead to incompetence of the Eustachian valve and thus allow of tricuspid regurgitation into the liver and the physical signs to which this gives rise, p. 39.

II. Of the superior vena cava.

The only definite sign associated with this condition is the superior vena cava murmur which may give valuable evidence as to the presence of tricuspid regurgitation, pp. 44, 218.

III. Of the great veins.

(*a*) The closure of the venous valves when due to tricuspid regurgitation may very occasionally give rise to a definite sound, p. 220.

(*b*) The incompetence of the valves not infrequently gives rise to a systolic murmur, p. 219, (291).
(*c*) The over-distension of the veins may give rise to pulsation in the supra-clavicular fossæ or the episternal notch, p. 134.
(*d*) Incompetence of the venous valves will give rise to distension of, and systolic pulsation in, the veins of the neck, pp. 39, **137**.
(*e*) When the azygos vein is over-distended as a result of tricuspid incompetence this may show clinically by distension of some of the intercostal veins, also any sudden increase of intrathoracic pressure—such as that due to coughing—may cause a recognisable impulse in one or more of them.

Clinical phenomena showing the condition of the auriculo-venous reservoir.

Direct study of the condition of the reservoir. When normal there is only one point at which the condition of the reservoir can be directly studied clinically—namely, at the point where the external jugular vein opens into the subclavian. The limiting valve is a short distance up the vein, and the portion on the proximal side of the valve is within reach of the finger—if pressed well down behind the clavicle. This portion of the vein is called by Sir James Mackenzie the jugular bulb, p. 133.

Pulsation in jugular bulb a true venous pulse, p. 134.

Its importance, pp. 39, **132,** 137.

Its fullness or emptiness shows the fullness of the auriculo-venous reservoir, p. 134.

Its tension shows the tension of the blood in the reservoir; a low tension being suggestive of heart weakness.

The volume of the pulsation in excess of normal shows the amount of tricuspid leakage.

The force of the pulsation is a most important guide to the strength of the right ventricle, pp. (88), 133.

Jugular bulb pulsation a surer guide to the strength of the right ventricle in later life than the arterial pulse is to that of the left ventricle, p. 134.

When abnormally full the distended veins become palpable and dilatation and pulsation of the neck veins will occur.

The condition of the auriculo-venous reservoir can also be studied indirectly.

The indirect study of the state of the reservoir is possible, because when overfull its distension is as a rule associated with overfullness of the right heart and of the lungs, and when unduly empty its underfilling is associated with relative emptiness of the heart and of the lungs.

This increase or decrease in the volume of the intrathoracic contents can be detected clinically by observing the state of the two non-rigid portions of its walls—namely, the floor as constituted by the diaphragm, and the upper boundary which consists of the soft tissues in the supra-clavicular fossæ, p. 163.

When the tissues of the diaphragm are sufficiently yielding and elastic (as in the earlier decades of life) an alteration in its level will provide adequate compensation for variations in the volume of the thoracic contents, p. 94.

When, as in later life its tissues are more rigid, evidence as to the fullness or emptiness of the reservoir is also given by fullness or hollowing above the clavicles, p. 94.

Fullness of the reservoir, such as results from cardiac failure, accompanied by tricuspid regurgitation, will show clinically, when the tissues are soft and resilient, by lowness of both sides of the diaphragm—namely, lowness of the upper level of the liver dullness on the right and of the gastric resonance on the left, with downward displacement of the heart, pp. (318), 363, (364), (366).

And to a less extent by filling-up of the supra-clavicular hollows.

When tissues are more rigid, there will be less alteration in level of diaphragm and more filling of supra-clavicular hollows, pp. 94, 134.

Emptiness of the reservoir (in common with the vessels of the lungs) will occur whenever there is compensatory lessening of the amount of blood in circulation (see 'Blood Volume, Compensatory Lessening of' p. 577), p. 57.

This will be shown clinically by the signs of diminution in the size of the intrathoracic cavity, which are summarised, at p. 579.

SUMMARY AND INDEX OF ARGUMENTS AND FACTS RELATING TO COMPENSATORY PHENOMENA IN CONNECTION WITH THE HEART AND BLOOD-VESSELS

IN CONNECTION WITH THE GREAT VEINS

The auricle and great veins constitute a compensation reservoir for the right ventricle, p. 2.

The tonic and contractile activity of the walls of the auricle and veins by tending to check tricuspid regurgitation help to compensate for incompetence of the tricuspid valve, pp. 13, 37, 133, 509, 521.

The inefficiency of the valves in the azygos vein and its branches enables it, in all probability, to act as a safety-valve-like outlet to the auriculo-venous reservoir, whenever there is a sudden tendency to its over-distension with blood or to the blood pressure in it being unduly raised—as in the act of coughing.

IN CONNECTION WITH THE BLOOD-VESSELS

In extreme myocardial weakness there is compensatory contraction of the peripheral arterioles so as to maintain the low blood pressure at its maximal height throughout the diastole, 59.

Weak pulse of high tension in enteric fever, p. 59.

In extreme heart weakness there is compensatory contraction of the peripheral vessels so as to give a maximal supply of blood to important organs, p. 109.

Pallor of the face in fainting, and in some cases of defective compensation in valvular disease, p. 450.

In myocardial weakness there is compensatory diminution in the amount of blood in the peripheral circulation. Case demonstrating this, p. (140).

When there is great limitation in the amount of blood available for the left ventricle (e.g. where there is fatty degeneration of the right ventricle and the left one is strong) there is compensatory increase in the force and the suddenness of the pulse wave, p. (156).

Strong collapsing pulse, showing fatty degeneration of the right ventricle with a strong left ventricle, p. (155).

Compensatory rise in the blood pressure where, from disease of the coronary arteries or other cause, a normal blood pressure does not suffice adequately to nourish the heart muscle, p. 144.

Characters of compensatory rise in the blood pressure as shown by the sphygmo-oscillometer, p. 144.

COMPENSATORY DIMINUTION IN THE AMOUNT OF BLOOD IN ACTIVE CIRCULATION IN HEART WEAKNESS. (See special summary, p. 577.)

COMPENSATORY PHENOMENA CONNECTED WITH THE HEART

In overstrain of a ventricle the stretching of the auriculo-ventricular orifice allows regurgitation to take place and thus checks the tendency to further dilatation of the ventricle, p. 32.

In myocardial weakness the inefficiency of the ventricular contraction results in a compensatory diminution in the amount of blood in active circulation, pp. **50**, 180, **185**.

When (as in adolescence) the tissues of the heart are relatively elastic, muscular weakness is accompanied by a compensatory diminution in the filling of the heart, p. 53.

This causes a diminution in the size of the heart, as observed clinically. A condition which may be called 'heart failure with small heart,' p. **54**.

COMPENSATORY PHENOMENA IN CONNECTION WITH THE RIGHT VENTRICLE

Compensatory overaction of the right heart often constitutes the first clinical sign that the left ventricle is being overtaxed, pp. 2, 46.

In valvular disease of the left side the amount of compensatory overaction of the right ventricle is a measure of the extent to which the left ventricle is failing to compensate for the valvular defect, pp. 47, **422**.

COMPENSATORY PHENOMENA CONNECTED WITH THE LEFT VENTRICLE

Compensation for valvular regurgitation. Its nature.

Dilatation and hypertrophy commensurable with the amount of valvular leakage are necessary, pp. 389, **412**.

Importance of cardiac distensibility in admitting of adequate compensatory dilatation, pp. 11, 150.

Softening effect of rheumatism upon the heart is of importance in aiding the development of compensation, pp. 11, 343.

Rigidity of the heart wall a cause of faulty compensation, p. 452.

Adherent pericardium interfering with compensation, pp. 397, (433), (455).

Relative rigidity of the heart in later life interfering with compensation for valvular disease, p. (456).

Pallor a sign of faulty compensation, pp. 450, &c.

Cardiolysis a remedy for the imperfect compensation that may result from adherent pericardium, p. 457.

COMPENSATION IN AORTIC REGURGITATION, p. 347

Compensatory diminution in the amount of aspiration is shown by weakness of the first sound and by cardiographic evidence, p. 409.

COMPENSATION IN MITRAL REGURGITATION

When perfect it depends on the left ventricle alone not on the right, p. 412.

Extra work is only thrown on the right side of the heart when compensation, from any cause, is imperfect, pp. 389, 422.

The ventricle develops sufficient hypertrophy and dilatation to compensate for the valvular leakage, p. 389.

The hypertrophy and dilatation necessary for the proper emptying of the ventricle is also adequate for its proper filling, pp. 389, 412, 417.

No extra work is thrown on the right ventricle when compensation is perfect, pp. 417, (422).

Pulmonary second sound not increased in loudness, pp. 389, (422), (424).

No dilatation of the left auricle, pp. 402, 417.

Development of compensatory dilatation of the ventricle by means of its own aspiratory force, p. 397.

Cases demonstrating the increased aspiratory force which the ventricle *can* develop in mitral regurgitation, p. (433).

Expansion phase well marked in the cardiogram, pp. 385, 403, 440, Plate XII.

Evidences of overaction of the right ventricle, which are characteristic of failing compensation, disappear when it becomes perfect, p. (423).

Cases showing great dilatation of the left auricle where compensation was imperfect, pp. 433, 455.

Compensation in Mitral Stenosis

The left ventricle alters its mode of contraction so as to increase its aspiratory power both muscular and mechanical, pp. 396, 405, 418, (423).

Its contractile force is concentrated at the end of systole, pp. 390, 419.

The characteristic sharp ending of the first sound in stenosis is thereby explained, pp. 390, 418.

Its contractile force is increased in order to increase at the same time its expansile force, pp. 405, 418.

The first sound is therefore abnormally loud, pp. 390, 405, 418, (423), (425).

Failure of compensation or temporary embarrassment increases the loudness of the first sound very greatly and it lessens as compensation improves, pp. 419, (423), (425).

This increased force explains the hypertrophy of the left ventricle which is sometimes recognisable in mitral stenosis, pp. 391, 418.

Evidence of increased aspiration in mitral stenosis is given of the cardiograph, p. 406.

'Suction recoil' wave in the cardiogram, pp. 407, 428, (432).

The increased aspiratory force thus developed compensates for the narrowing of the mitral orifice, and there is no hindrance to the circulation through the lungs, pp. 391, 419.

The left auricle is often found to be normal in cases dying without loss of compensation, pp. 391, 419.

The right ventricle does not have extra work thrown upon it in compensated mitral stenosis, pp. 406, 420.

The true loudness of the pulmonary second sound is not greater than normal in compensated mitral stenosis, pp. 391, 420, (425), (426).

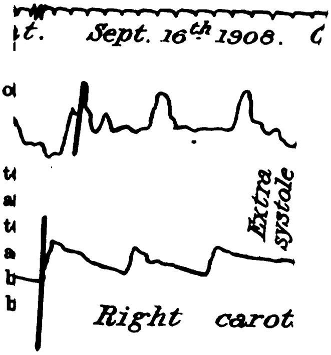
When compensation is imperfect the second sound is accentuated, but lessens in loudness as compensation improves, pp. 420, (422).

The well-known 'accentuated second sound' of mitral stenosis is a 'spurious accentuation' due to the right ventricle adopting the same type of forcible beat which the left ventricle has to adopt, pp. 392, 423, and 424 footnotes.

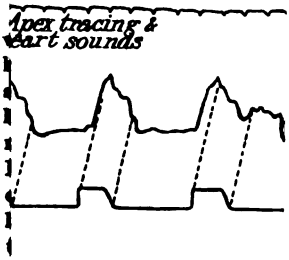
Pulmonary second sound much louder in anæmia or uncompensated valvular disease than in compensated mitral stenosis, pp. 392, 415, (421).

PLATE IX

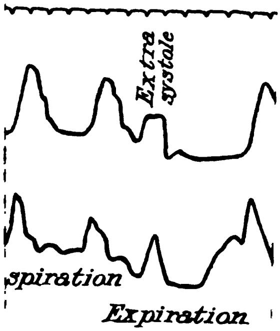
Tracing No. 1



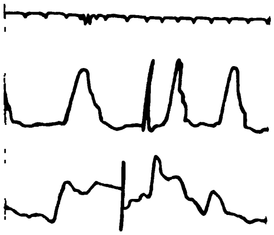
Tracing No. 2



Tracing No. 3



Tracing No. 4



SUBJECT INDEX

Figures in black type refer to important references.
Figures in small type refer to ordinary references.
Figures in parentheses (8) refer to references to clinical cases.
Figures above 532 refer to the Summary.

'ABSOLUTE' overstrain, 310, 342
Absolute percussion dullness, definition of term, &c., 21
,, ,, of heart, upward increase of, in anæmia, &c., 25, 228
,, ,, disappearance of, in myocardial weakness, 56, (119)
,, ,, of liver: its value as a guide to the amount of blood in circulation, **185**
,, ,, ,, its decrease due to altered conductivity of the liver tissue when relaxed, 191
,, ,, ,, its diminution a valuable sign of heart weakness, 195
Accentuation of the first sound of the heart in mitral stenosis a compensatory phenomenon, 390, 425
,, of the right ventricle first sound an evidence of failure of the left ventricle, 47
,, of the aortic second sound in disease of the mitral valve due to increased aspiration, 418, 423
,, of the pulmonary second sound in anæmia, 244, 337, 415, **421**
,, ,, ,, ,, ,, in failing compensation for valvular disease, 422
,, ,, ,, ,, ,, in compensated mitral stenosis is really a false accentuation, 392, 423, 424
Adherent pericardium: physical signs of, 454
,, ,, a cause of defective compensation in mitral regurgitation, (433), (454), (455)
Adhesions limiting the distensibility of the right ventricle a cause of sudden death, especially under anæsthesia, 62
Adolescence, softness of the fibrous tissues in, 8, 10, 535
,, ,, ,, ,, in relation to cardiac enlargement, 8, 10
,, ,, ,, ,, in relation to diminution in the size of the heart, 50, 54, 537, 567
,, ,, ,, ,, in relation to rise of diaphragm in compensatory diminution of the blood volume, 72, 94
,, early diagnosis of cardiac failure in, 28
Adolescent type of cardiac dilatation, **12**, 211, **330**, 543
,, ,, pathological aspects, 247, 546
,, ,, clinical aspects, 547
,, ,, in anæmia, 228
,, ,, in cardiac overstrain, 309
Adult type of dilatation of the right ventricle, 10, **31**, 330, 341, 557

ANÆMIA AND HEART FAILURE.
  Various relationships of heart failure to anæmia, 227, 448
  Anæmia and pallor compared, 450
  Primary anæmia of adolescence distinguishable from secondary anæmia by its type of cardiac dilatation, 243

ANÆMIA OF ADOLESCENCE, heart failure in, **227**
  Characteristic dilatation upwards and to the left, 228
    ,, ,, due to dilatation of the right ventricle, 232
  Diversity in shape of the dilated heart, **289**
  Dilatation towards the right infrequent, 238, 354
  Factors determining the type and the amount of the dilatation, 241, 289
    Overstrain of right ventricle, 243
    Age, 239, 349
    Cardiac malnutrition, 242
    Left ventricular failure not a factor, 240
  Left ventricular failure as a rule infrequent in anæmia, 240
    ,, ,, ,, sometimes extreme, 281
  Tricuspid murmur infrequent in anæmia, 239
  Pulmonary artery murmur in, 26, 211, **254**, 334
  Mid-diastolic murmur and third sound arising in right ventricle, **282**, 341

Aneurysm of the transverse arch of the aorta:
  Its diagnosis from upward dilatation of the right ventricle, (213)
  A cause of displacement of the heart, 345
Aneurysm, intra-pericardial and displacement of the heart, (345)
Aneurysmal dilatation of the pulmonary artery, the cause of the pulmonary systolic murmur, 26, 253
Angina pectoris rare in adolescence and early adult life, 82
  Increasingly frequent as age advances, owing to increasing amount of mechanical aspiration, 36, 82, 126, 567
  Angina pectoris of vasomotor origin due to colitis, (127), (130)
Aorta, its first part constitutes an expansion chamber for the left ventricle:
  Rigidity of its first part, a hindrance to the circulation, 74
    A cause of difficulties in diagnosis, 74, (109)
  Compression of the superior vena cava by the systolic distension of its first part, the cause of the ventricular wave in the venous pulse, 512, 531, 588
Aortic 'recoil waves' in the ventricular cardiogram, 501, 512
Aortic regurgitation, principles of compensation in, 347
  Accompanied by compensatory lessening in the amount of ventricular aspiration, 409
  Accompanied by downward displacement of the heart, 347
  Pallor a sign of defective compensation in, 451
  Lessened dilatation and hypertrophy a sign of the onset of myocardial weakness in, (112)
  Presystolic murmur of, usually a mid-diastolic murmur, 394
Aortic regurgitant murmur, an 'expansion murmur,' 399
  ,, ,, mid-diastolic addition to, 394
Aortic second sound accentuated in mitral stenosis, 418, 423
Aortic systolic murmur:
  Its conduction to the back, 444
  Differential diagnosis from an 'arterial compression' murmur, 226
    From a pulmonary artery murmur, 215
    From a superior vena cava murmur, 218

APEX OF THE HEART.
  Displacement upwards of the apex of the heart in the adolescent type of dilatation of the right ventricle, 19, 234, **348**, (360), &c.
    Statistics showing its frequency in the heart failure of anæmia, 349

APEX OF THE HEART.—Upward displacement of (*continued*).
  Statistics showing its relationship to the amount of upward dilatation, 352
    Showing that with any given amount of dilatation the frequency of elevation increases with age, 350
    Showing that its frequency is greater in patients twenty to twenty-five years of age than in younger patients, 350
    Showing that its frequency increases proportionally to the amount of broadening of the right ventricle towards the left, 352
    Showing the absence of any dilatation to the right, when the apex is high, 354
    Showing the absence of a tricuspid murmur when the apex is high, 355
    Showing the absence of any signs of left ventricular failure when the apex is high, 356
  The size of the left ventricle in relation to the elevation of the apex, 357
  Diminution in the size of the left ventricle a probable factor in its causation, 354
  Stretching of the anterior wall of the right ventricle, as a whole, a factor in its causation, 357
  Summary of the agencies at work in its causation, 359
  Fixation points of the heart in relation to the upward displacement of the apex, 358
  Increase of the right ventricle towards the left is the actual cause of the elevation of the apex, 359
  The apex may sometimes be elevated by downward enlargement of the right ventricle, 348
  Upward displacement in patient aged sixty-two, (83)
  Displaced downwards in aortic regurgitation, 346
    In chronic venous congestion of the lungs, 363, (364)
  Variation in its level with change of position, 235, (311), (312)

Apex beat weak or absent in myocardial weakness, 56, (68), &c.
'Arterial compression murmur,' arising in subclavian artery in anæmia, 226, 281
  Its diagnosis from an aortic systolic murmur, 226
  ,, ,, from a venous valve murmur, (291)
  Its disappearance on moving the shoulder, 226
  Its artificial production by the pressure of the stethoscope, 226
Arterial pulse:
  Weak pulse of high tension diagnostic of myocardial weakness, e.g. in enteric fever, &c., 59
  Normal or strong pulse in adult and later life an uncertain guide to the state of the heart and circulation, 35, 143
  Collapsing high pressure pulse in fatty right ventricle with strong left ventricle, (155)
Arterial resistance:
  Its amount difficult to estimate in later life, 74, 142
  Its bearing upon a failing heart difficult to estimate in later life, 142
  Increased arterial resistance sometimes a compensatory phenomenon, 144
Arterioles, peripheral, compensatorily contracted in great heart weakness, 59
Aspiration by the ventricles of the heart due to transverse contraction of muscular fibres of its wall, 384, 400, 412
  In adolescence due to muscular expansion only, 52
  In adult and later life mechanical elastic recoil takes an increasing share in the filling of the ventricles, 53, 78, 79
  Lessened expansion in muscular weakness causes lessened filling of the heart, 54
    And compensatory diminution in the volume of the blood in circulation, 55
  Its compensatory increase in mitral stenosis, 390, 412
    ,, ,, ,, ,, regurgitation, 389, 412
    ,, ,, lessening in aortic regurgitation, 409

Aspiration by auricles after their systole, 508, 512 (point 2), 530

Asystole—risk of death from—does not occur in adolescence because of compensatory underfilling of the ventricles, 53, 82
  Increasing risk of death from it as age advances, owing to increasing amount of mechanical aspiration, 53, 82, 567

AURICLES.
  Their muscular walls are sensitive to mechanical stimulation by changes in blood pressure, 504, 507
    By their tonic activity they endeavour to maintain a constant blood pressure within the auricle, 503
    A definite contraction may result from—
      1. Nodal stimulation, as in the case of the auricular systole, 529, 585
      2. From a sudden increase of pressure, as in tricuspid regurgitation, 504
      3. Probably also from a sudden lessening of pressure, as during ventricular aspiration, 528, 586
      4. A prolonged contraction appears to result from free tricuspid regurgitation, 509, 521
  Their functions, 387, 528
    1. To act as a reservoir of supply to the ventricle, 387
    2. By their contraction (' systole ') to close and stretch the auriculo-ventricular valves prior to the ventricular systole, 388
    3. To precipitate the ventricular systole by the mechanical stimulus which results from the sudden stretching of the ventricular walls by the inrush of blood from the auricle during the auricular systole, 530, 585
    4. By their tonic and contractile activity to help to check regurgitation through the auriculo-ventricular valves, 37, 133, 509, 521
    5. By their tonic activity to maintain in the ventricle during the relaxation phase the blood which was aspirated into it during the expansion phase, 528, 587
  Left auricle:
    Not very accessible to clinical examination, 47
    When dilated causes increased conduction of heart sounds to left interscapular region, 47, 444
    When much dilated gives a dull percussion area in left interscapular region, 47
    Great dilatation in mitral disease with failing compensation, 435, 455
    Little or no dilatation in mitral disease when compensation is good, 402
  Its appendix does not approach anterior chest wall, 231
  Its proximity to the vertebræ admits of the conduction of a mitral regurgitant murmur down the spine, 443
  Right auricle:
    When dilated it does not usually approach the anterior chest wall, 41
    Pulsation due to its dilatation very rarely observable, 41
    When dilated it may cause increased conduction of the cardiac sounds in the lower dorsal region to the right of the spine, 42
    Case showing extreme dilatation, (210), (339)

Auricular appendix, its functions, 388, 530, 585

Auriculo-venous reservoir, 584

Auriculo-ventricular valves, possibility of their closure at the end of the expansion phase, 388

Austin Flint murmur, 394

BINAURAL stethoscope, theory of sound conduction by it, 282
  Light application necessary to hear a third sound of functional origin in right or left ventricle, 282

Blood Pressure:
  If high, there is a varied relationship to cardiac failure.
    (*a*) Due to toxic arterial resistance and throwing extra work on the heart.
  High blood pressure from the absorption of toxins in pneumonia, (103)
    (*b*) Compensatory in character and necessary for the adequate nutritition of the heart, 144
    (*c*) Compensatory in character, but insufficient (owing to myocardial weakness) for the adequate nutrition of the heart—compensatory distinguishable from non-compensatory by means of the sphygmo-oscillometer, 144
  Pathological variations in, a source of diagnostic difficulties in adult and later life, 142
  In the pulmonary artery, a factor in the causation of the pulmonary systolic murmur, 271, **555**
    Case where this was the main factor, (274), **300**
Blood Volume: its Compensatory Diminution, 577
  In deficiency of essential constituents of the blood, 163, 169
    In starvation in enteric fever, 168
    In treatment of gastric ulcer, 169
    In hæmorrhage, cholera, &c., 173
    In neurasthenia, &c., 177
  In heart weakness owing to the heart's inability to keep the normal volume in circulation, **54**, 180, 185, **195**, 578
    Its occurrence of great diagnostic value in later life, 94
  Clinical evidences of its compensatory diminution, 579
    Rise in the level of the diaphragm, **163**
    Hollowing of supra-clavicular fossæ, 94, 134, 139
    Diminution in the area of the liver dullness, **185**
    Smallness of jugular bulb pulsation, 97, 133, 137, **583**
    Emptiness of the veins, 57, 61, 95, **132**, 187
    Case proving the compensatory nature of the emptiness of the veins in myocardial weakness, (140)
Breathlessness:
  In heart failure of adolescence, 12
    In the right ventricular dilatation of anæmia, 228
    Its cause, 245
    Characteristic sighing respirations, 13
    'The deep breath,' its rationale, 15
  In adult life, its greater importance, 36
  In later life, diagnostic difficulties, 113
Bruit de diable:
  Due to constriction of dilated veins, **15**, 337
  Its characters, 15, 337
  Its variation in loudness with both respiration and cardiac action, 16
  Its diminution or disappearance with the recumbent posture, **285**, 337
Bruit de galop in right ventricular dilatation, due to the occurrence of a third sound, 340

Calf muscles, pains in on exertion, a sign of myocardial weakness, 467, 470, (471)
  See Leg Pains
Cardiac cycle consists of three phases: ventricular contraction, expansion, and relaxation, 387
  Measurements of the three phases, 428
  See Sounds of the Heart
Cardiac dilatation, *see* Dilatation

Cardiac dullness:
Method of percussion, 20 to 25
Its shape in heart failure in adolescence, 287
,, ,, ,, adult life, 40
Its increase upwards and to the left in the adolescent type of right ventricular dilatation, 25, 232
Its increase on lying down in anæmic heart failure, 266
Its diminution or absence, a sign of myocardial weakness, 54, 56, (68)
When not increased in heart failure the cause may be weak muscle or abnormal rigidity of the heart wall, 34, 86, 91, 147
Cardiac failure (*see* Heart Failure), 7
Cardiac impulse (*see* Apex Beat)
Its character in adolescent dilatation of the right ventricle, 17, 231, 331
Diagnostic value of impulse in outer part of third left interspace, 19
,, ,, at sternal end of fourth left interspace, 40
If weak or absent, a sign of myocardial weakness, 60
Its disappearance, after being abnormally increased, a sign of improvement,
,, ,, ,, ,, a sign of myocardial weakness, (105)
Cardiogram:
Method of its interpretation, 400, 494, 510
In aortic regurgitation, 409
,, mitral ,, 403
,, ,, stenosis, 406
Cardiographic evidence as to the nature of the expansion phase of the cardiac cycle, 400
,, ,, of increased aspiration in mitral regurgitation, 403
,, ,, ,, ,, ,, stenosis, 406
,, ,, ,, lessened aspiration in aortic regurgitation, 409
,, ,, concerning the third sound of the heart, 427
,, ,, ,, mitral mid-diastolic murmur, 408
Cardiographic evidence as to the occurrence of ventricular extra systoles owing to mechanical stimulation of the ventricle, 497
,, ,, ,, tonic and contractile activity of the walls of the auriculo-venous reservoir, **500**, 504, 506
,, ,, of value in the diagnosis of aneurysm of the transverse and of the aorta, 215
Cardiolysis in defective compensatory dilatation, 457
Cardio-respiratory murmur: its diagnosis from a mitral systolic murmur, 49, 446
Classification of types of cardiac failure, 6
Coldness on exertion a sign of myocardial weakness, (365)
,, ,, due to faulty circulation in the muscles, 472
Collier, Dr. Mayo, on the functions of the auricles, 388
Colon dyspepsia a cause of palpitation, 119
,, ,, ,, vasomotor angina, (127), (130)
,, ,, ,, local asphyxia, (128)
,, ,, simulated by pain due to dilated thigh veins, 465
Compensatory phenomena in connection with the heart and circulation, *see* Summary, p. 592
Compensatory diminution in the volume of the blood in circulation, *see* Summary, p. 577
Compensatory rise in the diaphragm, **163**
,, diminution in the size of the more elastic vascular viscera, 163, 178
,, diminution in the size of the heart in myocardial weakness, **50**, 186

Compensatory emptiness of the veins in heart weakness, case proving it, (140)
,, dilatation of the left ventricle in mitral disease by means of its own aspiratory force, 397, 416, 436
Compensatory character of muscular cramp for improving the nutrition of exhausted muscles, 477
Conductivity of the liver tissue to percussion vibrations, pathological variations in, 24, 191
Conus arteriosus of the right ventricle:
  Its distensibility in adolescence, 4, 9
  ,, ,, especially in anæmia, 228
  Cases showing dilatation confined to it, 292, (293), (294)
  Extreme strain upon the conus during systole, 10
  Its special distensibility lessened in adult life, 31
  ,, ,, usually lost in later life, 32
Cramp on exertion a sign of faulty muscular nutrition, 467, 471, (477)
Cyanosis, 37, (469)

Davies, Dr., on circulation of blood through diseased hearts, 389, 451
Deep percussion dullness, *see* Relative Dullness, 21

Diagnostic Difficulties:
  Connected with the radial pulse in adult later life, 35
  Case where a normal pulse was really one showing heart failure, (35)
  The interpretation of a high blood-pressure. Is it too high? Is it normal for the patient? Is it too low?—142
  False and true venous pulsation in the neck veins, 14, 39
  The increase and decrease of pulsation in the neck veins in heart failure, table of phenomena, 137
  Dilatation of the right ventricle, Essay VII, 328
  Pulsation in the second left interspace and aortic aneurysm, 211, (213)
  Pulmonary systolic and aortic systolic murmur, 215
  ,, ,, ,, tricuspid systolic murmur, 259, 279
  The murmurs due to tricuspid regurgitation, 217
    The tricuspid systolic, 44, 217
    The superior vena cava murmur, 44, 218
    The venous valve murmur, 219, (291)
  The arterial compression murmur, 226
  Aortic systolic murmur, 45
  ,, regurgitant murmur when there is a mid-diastolic addition to it simulating a true Austin Flint murmur, 394
  The mitral regurgitant murmur, 48
    Estimation of the amount of regurgitation by the loudness of its conduction down the spine, 442
    And cardio-respiratory murmurs, 49, 446
  In connection with the recognition of heart failure in later life, 110
    Summary of opposing statements, 160
  The estimation of the distensibility of the heart wall in later life, 147

Diagnosis of cardiac enlargement from displacement, 151
  Displacement downwards simulating enlargement, (266), (365)
  ,, to the left ,, ,, (374)
Diagnostic value of slight lateral displacement of the heart in disease of the lung, (375), (376)
Diagnostic value of leg pains and of dyspnœa respectively in cardiac failure, 469
Diagnostic difficulties in connection with pallor and anæmia in heart disease, 450, 453, (454)

Diagnostic difficulties associated with the recognition of referred abdominal pain due to dilatation of the deep veins of the thigh, (463), 465

DIAPHRAGM:
  Its normal level, 163
  Its degree of arching determinable by percussion, 22, 165
  Its elevation by pathological changes in the lung, 166, (167)
      „ „ diminution in the amount of blood circulating in the thoracic viscera, 261, 580
    In malnutrition: in enteric fever, 168
      „ in gastric ulcer, 171
    In cardiac weakness, 57, 180
    In neurasthenia, &c., 177
  Low in emphysema, 362
    „ in pleuritic effusion, &c., 363
    „ in chronic venous congestion in valvular disease, &c., (318), 363, (365), (366), 591
  Diastolic wave in the neck veins, a sign of rigidity of the right ventricle, 398

DILATATION OF THE HEART AND VEINS

Theory of dilatation of the ventricles when over-strained in any way, 6, 9, 31, 211
Factors which determine the type and the amount of dilatation, 542
Various types of dilatation, 543
  Adult and adolescent types of dilatation compared, 5, 10, 31, 330
  Relative liability to dilatation of the right and left ventricles respectively at different periods of life, 10, 31, 46, 309
    with different kinds of exertion, 308
Of the right ventricle, 329
  Upwards and to the left in adolescence, 4, 10, 25; 228
    Clinical aspects of, 14, 228, 330
    Early recognition of, 28
    Various types of, in anæmia, 289
    Its relationship to the pulmonary artery murmur, 264
    Characteristic form of dilatation in overstrain, 309
      Cases showing great dilatation, (235), (304)
  To the right in adult life, 37
  Lessened frequency of dilatation in later life, 5, 73, 79
Left ventricle less liable to be affected by overstrain in adolescence than in adult life, 46, 308
In compensation for valvular disease, *see also* Ventricle, Left, 397
Of the right auricle (*see also* Auricle), 41
Of the superior vena cava, a cause of a murmur, 44, 218, (224), (280)
Cases showing extreme dilatation of the heart:
  Cases in adolescence, (7), (277), (281), (304), (314)
    „ „ later life, (320)
    „ of the left auricle, (455)
    „ „ right auricle, (236)
Does not occur in great myocardial weakness, 6
Dilatation of a weak heart a sign of returning strength, 63, (102), (294), (304), (316), 570
Dilatation of the heart simulated by displacement:
  1. Downwards, owing to lowness of the diaphragm, 362
    In emphysema, 362
    In chronic venous congestion of the lungs, 363, (364), (366)
  2. Downwards, owing to elongation of the first part of the aorta in aortic regurgitation, 346
  3. To the left, in collapse of the left lung, (374)

DISPLACEMENT OF THE HEART, ESSAY, 345
The relative mobility of the heart, especially in adolescence, (371), (377)
  Rapidly developed displacement from collapse of lung, (371)
  Mobility of the tissues forming the posterior mediastinum, 379
  The fixation points of the heart are only relatively 'fixed,' 371
  The main fixation points, 379
    Inferior cava where it passes through the diaphragm an important one, 380
    Their bearing upon displacements in general, 379
      „ „ upward displacement of the apex, 358
      „ „ rotation of the heart in displacement to the right, 379
      „ „ the rotation of the right auricle towards the anterior chest wall, (381)
      „ „ rotation of the heart in displacement to the left, 383
Altered relationships of a displaced heart, 379
Murmurs due to cardiac displacement, 383
Causes of displacement may be intrinsic or extrinsic, 345
Intrinsic causes—
  Aneurysm, 345
  Intrapericardial aneurysm, (395)
  Aortic regurgitation a cause of downward displacement, 346
  Upward displacement of apex due to downward enlargement of right ventricle 348
    „ „ due to adolescent dilatation of the right ventricle, 348
    „ „ „ „ „ „ *See* Apex
Extrinsic causes of displacement of the heart, 361
  Forwards, 361
  Backwards, 361
  Downwards, due to lowness of diaphragm, 362
    Effusion of liquids into pleura, 362
    Over-distension of lungs with air, 362
    Over-distension of thoracic vessels with blood, 363
      In 'chronic venous congestion,' (318), (364), (366)
    Growth of tumours in the thorax, (379)
  Upward displacement, 368
    By adhesions, 371
    By excess of abdominal pressure, ascites, &c., 368
    By traction owing to diminution in the size of the thoracic viscera, 368
      In pulmonary fibrosis in phthisis, &c., (167)
    In compensatory diminution in the volume of blood in circulation, in defective nutrition, &c., 168, 368, (370)
    In heart weakness, 180, (368)
  Lateral displacement, 371
    Diagnostic value of small amount of lateral displacement, 375, (376)
    To the left, (371), 373
      „ „ simulating enlargement of the heart, (374)
    To the right, 377
      An extreme case, (377)
      Case due to cancer of lung, (379)

DISTENSIBILITY OF THE HEART WALL, *see* Summary, pp. 533 to 541
  Variation in the heart's distensibility with age, 535
    Great in adolescence, 5, 7, 9
    Lessening as age advances, 6, 10, 33
    Practically lost sometimes in later life, 6, 79, 81, 85

DISTENSIBILITY OF THE HEART WALL (*continued*):
  Local variation in distensibility—
    Great in adolescence, 8
    Local variations in the distensibility of the walls of the right ventricle, 4, 9
    Right ventricle more distensible than the left, 11
    In adult life local variations in distensibility are less, 5, 11, 31, 309
    ,, ,, ,, left ventricle as dilatable as the right, 31
  In relation to heart failure without enlargement, 6, 50, 86, 91, 537
  Its clinical estimation, 539
    Simple in adolescence, 54
    In adult life often not easy, 34
    In later life often extremely difficult, 147
Dropsy, not a sign of myocardial weakness, 94
Dullness, *see* Absolute, Relative, Cardiac Dullness, and Liver Dullness

ELASTICITY of the heart wall, *see* Distensibility, 7
Elastic thigh-supports for the cure of dilated deep thigh-veins, 466
Enlargement of the heart, *see* Dilatation and Heart Failure
Enteric fever, pulse characteristic of heart weakness in, 59
  ,, ,, diminution of blood volume in, 168
  ,, ,, where percussion dullness did not indicate the true size of an enlarged liver, 153
Expansile force of the blood aspirated by the left ventricle shown by the cardiograph, 436
Expansile tone of the colon, a cause of vasomotor angina, (127)

EXPANSION PHASE OF THE CARDIAC CYCLE, 384, 400

The ventricles enlarge suddenly after contraction in virtue of an active expansion of the muscular fibres, 384, 400
This expansion is due probably to a transverse contraction of the shortened fibres causing them to lengthen, 384, 400
The heart muscle is known to remain hard for an appreciable time after the closure of the semilunar valves, 385, 401
The cardiograph shows the ventricle to be enlarging immediately after the closure of the semilunar valves, 385, 403, 521
An expansion phase must be recognised in the cardiac cycle, 386, 428
  Three phases of the cycle: contraction, expansion, relaxation, 387
  It lasts one-tenth of a second, 386, 428
  Its duration does not vary materially as is the case with contraction, 387
  The duration of the expansion phase is adequate for the complete emptying of the auricle into the ventricle, 387
The ventricle is therefore a double-acting pump, filling itself with its own back stroke, 384, 400
The recognition of a muscular expansion phase explains the compensatory phenomena of valvular disease (*vide* Summary, Compensatory Phenomena, 592), 412
It explains the compensatory underfilling of a heart that is muscularly weak, 52
It explains the diminution in the size of the heart which characterises severe myocarditis when occurring in a 'distensible' heart, 53
Feeble muscular aspiration accounts for the compensatory diminution in the blood volume which is observable in myocardial weakness, 52, 80

Extra systoles, due to mechanical stimulation of the ventricle, 495, 497

FATTY degeneration of the right ventricle with a fairly strong left ventricle, (154), (156)
Fatty degeration, *see* Myocardial Weakness

FIBROUS TISSUES:
  Their immaturity in adolescence deprives the heart of any support they might give, 5, 10
  In adolescence the resisting power of the heart wall depends mainly upon its muscular strength, 9
  The heart in adolescence is very distensible, and increases or diminishes readily in size, 8, 53
    In adolescence, compensatory underfilling of the ventricle in myocardial weakness takes place readily, 54
  Their immaturity also allows changes in the size or position of organs to take place readily, 72
    Diaphragm rises readily when bulk of thoracic contents is diminished, 94
    Displacement of the heart takes place readily in diseases of the lung or pleura, (371)
  Their increasing strength as age advances gives support to the heart wall, 51, 53
  In adult and later life dilatation takes place less readily than is the case earlier in life, 31, 79
  In later life this change may amount to what is practically a fibroid degeneration of the heart wall itself, 5, 78
  This increase in the resisting power of the heart wall ensures its possession of mechanical elastic recoil after contraction in addition to its muscular expansile power, 53, 80
    This change lessens or prevents compensatory diminution in the size of the full ventricle in myocardial weakness, 80
    It lessens, but does not prevent compensatory diminution in the volume of blood in circulation in myocardial weakness, in adult and later life, 80
    It renders the occurrence of angina pectoris and of death from asystole possible in adult and later life, 54, 82
  Increased resisting power of fibrous tissues makes changes in the size and position of organs take place less readily, 72, 76, 94
    Changes in the level of the diaphragm take place less readily and are less extreme in adult and later life than in adolescence, 72

'The fidgets': restlessness caused by dilatation of the deep thigh-veins, 462
Filling of the ventricle, compensatorily lessened in extreme muscular weakness in distensible hearts, 53
  Compensatory underfilling less complete when the heart walls possess mechanical elastic recoil, 80
Fixation points of the heart, *see* Displacement of Heart
Foxwell, Dr., on Anæmic Heart Failure, 234
'Functional' heart murmurs:
  Pulmonary systolic, 254
  Mitral systolic, 48, (281)
  Mitral mid-diastolic murmur, (286)
  Tricuspid regurgitation, 43, 279
  Tricuspid mid-diastolic murmur, 46, 282

GASTRIC resonance, as an indication of the level of the diaphragm, 164
  Variations in its level of great diagnostic value in cases of myocardial weakness, 57, 94, 180.
  Reverberation as a method of its detection, 480
Gastric ulcer with extreme diminution of blood-volume and small liver dullness, (171)
Dr. Lockhart Gibson on the mitral mid-diastolic murmur, 384, 400
'Globus hystericus,' in dilatation of the right ventricle, 14, (212), 342

HABIT palpitation after severe myocardial weakness; after influenza and diphtheria, 117, (119)
Habit sluggishness of the vasomotor mechanism, a cause of intermittent claudication, 475, (476)
Habit tachycardia after overstrain, (324)
Hæmorrhage causing high diaphragm, (176)
„ „ small liver dullness, (203)
Hæmic murmur of anæmia, *see* Pulmonary Systolic Murmur
Hæmic murmur in the neck, *see* 'Bruit de diable,' Superior Vena Cava Murmur, Arterial Compression Murmurs, and Venous Valve Murmur

HEART FAILURE, definition of term, 1
  With enlargement, 542
    In the distensible heart of adolescence, 12
    In the moderately distensible heart of adult life, 35
    In the relatively rigid heart of later life, 70, 571
    In anæmia, 227
    In overstrain, 308
  Without enlargement, 50, 85, 564
    In the distensible heart of adolescence, 53, **565**, **567**
    In heart failure with small heart, 54
    In the moderately distensible heart of adult life, 53, **566**, **569**
    In moderately strong but relatively rigid hearts, 73, **86**, (87), (89)
    In weak and relatively rigid hearts, 91
    Complicating pneumonia, (97)
    But accompanied by signs of a normal heart and normal circulation, 149
Heart sounds, their measured loudness in health and disease, 420–426
  Method of measuring their loudness, 413
  See Sounds of the Heart
Heart wall, elasticity of, 7
„ „ distensibility of, *see* Distensibility, and Summary, 533
„ „ relatively rigid in later life, 5, **76**
High diaphragm a sign of compensatory diminution in the blood volume, **163**, 580

IMPULSE of heart, *see* Cardiac Impulse
Intermittent claudication, 471
  A sign of myocardial weakness, 92, **469**
  Due to vasomotor disturbance, 472
  Due to habit sluggishness of the vaso-dilator mechanism, (476)
  Cured by brisk exercise, 477
Interventricular septum, part played by it in the causation of the raised apex beat in adolescent dilatation of the right ventricle, 237, **359**
„ „ its position shown on the chest by the limitation of the area of audition of the third sound to the ventricle in which it arises, (284)
Intraventricular pressure must be the agent causing dilatation of the heart, 6, 31
„ „ a high pressure necessary for causing upward dilatation of the right ventricle in adolescence, 239

JUGULAR BULB, palpation of, its clinical value, 133, 583, 590

LATER life, early diagnosis of heart failure in, 70, 571

LEFT VENTRICLE (*see* also Ventricles), 561
  Dilatation and hypertrophy necessitated by compensation for valvular leakage, 47, 389
  Dilatation in mitral incompetence brought about by increased aspiratory force, 397

LEFT VENTRICLE (*continued*):
  Hypertrophy in mitral stenosis sometimes accompanies its increased aspiratory activity, 391, 418
  Inadequate compensatory dilatation a sign of (*a*) abnormal rigidity of its walls; (*b*) weakness of its muscle, (65), 150
  Diminution in the amount of compensatory dilatation a sign of the onset of myocardial weakness, (112), (316)
  In overstrain in adolescence, 11, 309
    Dilatation often prevented by the prior failure of the right ventricle, 47, 309
    Sometimes dilatation well marked, (277), (281)
In adult life dilatation of the left ventricle equals or exceeds, in frequence, that of the right, 46, 316
Case of extreme dilatation, (320)

LEG PAINS OF CIRCULATORY ORIGIN, 459
  Due to LOCAL abnormality in the circulation—
    Due to dilatation of the deep thigh-veins, 459
    Probable pathology, pressure on nerves, 460
    Occurrence of pain on standing or in the evening is characteristic, 461
    Nature of discomfort sometimes indescribable, 462
    Pain often referred or segmental in character, 462
    Sometimes localised in groin and simulating colon dyspepsia, 465
      ,, only a remote effect on nervous system and a potent cause of neurasthenia, 462
    'The fidgets,' if coming on in the evening, a characteristic result of dilated deep thigh-veins, 463
    Referred hip-pain simulating neuralgia, (463)
    Easily cured by elastic support to the thigh, (464), 466
    A simple support made from elastic webbing bandage, 466
    Neurasthenia cured by elastic thigh-supports, (464), (465)
    Due to deficient blood supply to the muscles, 467
    Intermittent claudication, 471
      ,, ,, due to vasomotor disturbance, 472, (473)
      ,, ,, due to habit sluggishness of vaso-dilator mechanism, 475, (476)
      ,, ,, cured by brisk exercises, (474), (476)
Leg pains due to deficiency of the GENERAL circulation, owing to heart weakness, 467, 469
  In muscular weakness from malnutrition the calf muscles are the first to suffer, 468
  Pain in calf muscles due to transient heart weakness, (468)
    ,, ,, a valuable sign of myocardial degeneration, 92, 468
  Diagnostic value of pains in the calf muscles and of dyspnœa respectively in heart failure, 470, (471)
  Coldness on exertion a sign of defective muscular nutrition and often associated with leg pains, (365), (474), (475)
  Cramp on exertion due to faulty nutrition of the muscles, 472, (477)

LIVER
Method of its percussion, 21, 189
Normal level of the upper border of its dullness, 165
Variability of the lower edge of its dullness, 165
Causes of variation in its area of dullness, 189, 581
  1. Its tissue when abnormally tense or abnormally flaccid appears to some extent to lose its power of masking the resonance of underlying organs, 24, 191
    In an enlarged and congested liver the absolute dullness does not come down as near to the edge of the liver as would be the case in a normal organ, 24, 191

LIVER (*continued*):
Case of enlarged liver in enteric fever where the lower border of absolute dullness was three inches away from the edge of the liver, 153
When less distended with blood than normal there is also increased conductivity to percussion vibrations causing a diminution of the liver dullness, 24, 191
In cardiac weakness this diminution varies with the strength of the heart, so exactly as to suggest a close connection, 193
Diminished liver dullness a sign of myocardial weakness, 58, 95, 185, &c.
2. Liver dullness may also be lessened by the drawing up of the liver under an abnormally high diaphragm, 165, 190
Rise in the lower edge of the liver dullness in a rise in the diaphragm owing to the absorption of pleuritic effusion, (103)
3. Diminution in the area of liver dullness owing to undue flaccidity allowing it to fall away from the anterior abdominal wall and the ribs, 58, 190
Case proving this, (205)
4. Actual shrinkage in size when underfilled with blood, must play some part in causing a lessening of its dullness, 188, 189
Diminution in the size of the liver dullness due to other causes than heart failure (*see* Summary, p. 578), 152, 202
Diminution in the size of a liver enlarged by chronic venous congestion a sign of myocardial weakness and not of a gain in strength, (63)

Local asphyxia and syncope of the fingers a reflex result of irritation of an irritable colon, (128)
Long breath in dilatation of the right ventricle, 12
Loudness of the heart-sounds, measurement of the, 413
„ of right ventricular sounds in absolute and relative overstrain respectively, 29
Lungs, diminution in the size of the healthy lung in pneumonia, a sign of dangerous myocardial weakness, 97

MECHANICAL elastic recoil possessed by the heart wall in adult and later life, **79**
Its presence lessens or prevents compensatory diminution in the size of the heart in myocardial weakness, 53, 80
Lessens but does not prevent compensatory diminution in the volume of blood in circulation, 81
Renders the occurrence of anginal pain and of death from asystole possible, 53, 81
'Menière's disease,' symptoms of, simulating heart failure, 121
„ „ hyperæsthesia of semicircular canals, a valuable sign of, 123
Micturition: danger of adopting erect attitude when heart is muscularly weak, 126
„ rationale of the squatting attitude adopted by natives of India, 126
MID-DIASTOLIC MURMUR, 393, 408
A 'Relaxation,' murmur occurring at the commencement of the relaxation phase, 293, 408
It occurs therefore at the end of the expansion phase one-tenth of a second after the second sound, 394, 408
It is produced by the aspiratory force developed during the expansion phase, 393
It is due to the vibration of the segments of the mitral valve in the blood stream flowing from the auricle into the ventricle, 393, 408
The valve flaps are only free to vibrate on the relaxation of the musculi papillares, 393, 408

MID-DIASTOLIC MURMUR (*continued*):
  The mid-diastolic murmur can only occur when the flow from the auricle into the ventricle is prolonged until the commencement of the relaxation phase, 431
  Causes of prolongation are:
    1. Stenosis of the auriculo-ventricular orifice, **394**, **431**
    2. Dilatation of the auricle to a greater extent than the ventricle, **435**
    3. Slight lessening of the aspiratory force through muscular malnutrition, as in anæmia, 282
    Mitral mid-diastolic of functional origin, (286)
    Tricuspid „ „ „ (289), 341
  Mid-diastolic addition to the aortic regurgitant murmur can be caused in the same manner, but by the regurgitating blood flowing over the anterior aspect of the anterior mitral cusp, 394
Mitral regurgitation, process of compensation in, 389, 403, 416, **594**
Mitral stenosis, process of compensation in, 390, 406, 418, **595**
Mitral systolic murmur:
  The first reliable sign of left ventricular failure, 48
  Diagnosis from a tricuspid systolic, 48, 217
    „ „ cardio-respiratory murmur, 49, 446
  Diagnostic and prognostic importance of noting the loudness of its conduction down the spine, **442**
  Reasons for its conduction to the angle of the scapula, 442
  Absent in spite of extreme dilatation of the left ventricle, (281), (320)
Mitral valve:
  Its segments held rigid during the expansion phase; therefore no vibration of them possible before the onset of relaxation, 393, 431
  No flow of blood through the valve in normal heart after the end of the expansion phase, and therefore there is no sound in the normal heart at the onset of relaxation, 393, 430
  See also Mid-diastolic Murmur, Mitral Regurgitation, Mitral Stenosis
Muscular cramp: a compensatory phenomenon to improve the nutrition of exhausted muscles, (477)
Muscular expansion of the heart (*see* Expansion) in relation to compensation for valvular disease, *vide* Essays IX, X, XI, and XII, and Summary, p. **594**
  In relation to compensatory diminution in the size of the heart, **52**
  Develops the force whereby compensatory dilatation of the left ventricle is brought about in mitral regurgitation, 397, 436
Muscular strength of the heart wall is the main factor in resisting dilatation in adolescence, 4, 8, **9**, 31
  Difficulty in distinguishing between muscular and fibrous strength of the heart wall in later life, 34, 85, 147
Myocardial weakness, signs of:
  In adolescence, diminution or disappearance of cardiac dullness, 56, 60, 96
    Feebleness of cardiac impulse, 56, 96
    Characteristic pulse, 35, 56, **59**, 95
    High diaphragm, 57, 163
    Small liver dullness, 185
  In adult and later life, 94
    Compensatory diminution in size of heart less marked as age advances, 72, 79
    Changes in level of diaphragm less extreme, 72, 94
    Rise in the level of the gastric resonance of value, 94
    Diminution of the area of liver dullness of value, 95
    Signs of compensatory diminution of the blood volume given by (*a*) hollowness above the clavicles, 94
    (*b*) By emptiness of the venous system, 95
  Signs of myocardial weakness given by variation in the amount of pulsation in the neck veins, 132, 137

NECK veins: diagnostic value of phenomena connected with them, 132, 137
  See Veins
Neuralgia of the hip simulated by dilated deep thigh-veins, (463)
Neurasthenia due to dilated deep thigh-veins cured by elastic thigh-supports, 462, 466
Nitroglycerine: its action in vasomotor angina due to colon dyspepsia, (130)
  „ causes a filling-up of the veins when there is compensatory emptiness of them, (140)
  „ cautions as to its use in weak and 'rigid' hearts, 148
Nocturnal occurrence of attacks of palpitation or of angina pectoris suggests their reflex colon origin, (116), (131)
Nutrition of the heart, its importance in cardiac failure, 5, 449
  „ „ a factor in the heart failure of anæmia, 242

OPTIC neuritis in anæmia, (297)
Overaction of right ventricle:
  Signs of in adolescence, 4, 12, 82
    „ „ adult life, 40
    „ „ valvular disease in relation to compensation, 414
  Of great value in estimating the amount of cardiac rigidity present in heart failure in later life, (87), (89)
Over-exertion, a factor in the cardiac dilatation of anæmia, 247, (303)
Over-stimulation of the heart: a danger to be avoided in myocardial weakness, 111

OVERSTRAIN OF THE HEART, 308

Apportionment of strain on right and left ventricles, 10, 46, 309, 545
Definition of terms 'Relative,' and 'Absolute,' 310, 342
Conditions causing overstrain, 545
  Overstrain in adolescence, (212), (310), (312), (313)
    „ in adult life, 316
    „ in young man with unusual symptoms, and result, (324)
    „ due to the sudden cessation of severe exertion, 327
    „ in later life (83), (320)
    „ „ before estimating its amount, allowance to be made for degree of rigidity of the heart-wall, 86, 147
Overstrain of the right ventricle the result of valvular disease, (339), (435), (455), &c.
Overstrain an important factor in the causation of the heart failure of anæmia, 244
Overstrain of the left ventricle in adolescence, (281)

PAIN in the groin, due to dilated deep thigh-veins, 465
  „ in the legs, of circulatory origin (*see* Leg Pains,) 459
  „ in upward dilatation of the right ventricle, 36, (212), (313)
  „ anginal pain, *see* Angina Pectoris
Pallor and anæmia, definition of terms, 448, 450
  In transient heart weakness, 450
  In serious heart weakness, 451
  Due to deficiency in the circulating blood in faulty compensation, 452
  In aortic regurgitation, 452
  In mitral regurgitation (454)
  Liability of mistaking pallor due to faulty compensation for anæmia, 453, (456)
  Due to undue rigidity of the heart-wall interfering with compensatory dilatation, 454, (455), (456)
  Prognosis in, 457
  Risk of sudden death in patients with pallor, 458

Palpitation, 114
  „ as a cardiac habit, 117
  „ when nocturnal its reflex origin in the colon is suggested, (116)
Pathological facts in connection with dilatation of the right ventricle, 233, **247**

Percussion of the Heart, method, 20
  Nomenclature. The terms 'relative' and 'absolute' dullness preferable to 'superficial' and 'deep,' 21
  Value of firm and light percussion, 20, 22, 24
  Supposed unreliability of relative dullness, **20**
  Confirmation of relative dullness by percussing again with the lungs emptied, 22

Percussion of the Liver, 22, 189
  Degree of arching of diaphragm shown by percussion of its upper border, 23
  Percussion of its lower border usually neglected, 23
  Uncertainties associated with its percussion, 24
  Diminution in the area of absolute liver dullness due to changes in the conductivity of the liver tissue to percussion vibrations, 191
  Increased conductivity due to the increased tension which results from over-distension with blood, 24, 153
  Increased conductivity also probably due to the lessened tension which results from relative emptiness of its blood-vessels, 191
Generalisation as to firm and gentle percussion when a solid organ is overlaid or underlaid by a resonant one, 23
Points to be noted in percussion of the sternum, 25
Pericardium:
  Its power of limiting cardiac dilatation, slight in adolescence, increasing with age, 5, 11
  Its relationship to the superior vena cava, giving rise to the superior vena cava murmur, 44
Phear, Dr. A., on the third sound of the heart, 427
Physical signs of heart failure: their *absence* in pneumonia, &c., may show dangerous heart weakness, (97), (105)
Pneumonia, accompanied by a rapid dilatation of the right ventricle and a rise in the apex beat, (360)
  High diaphragm in, showing myocardial weakness, (97), (224)
  Interpretation of phenomena due to cardiac weakness, 98, 105
Pneumococcal toxin, causing a rise in the blood pressure, 103
Position, change of, affecting:
  Amount of upward dilatation of the right ventricle, **266**
  'Bruit de Diable,' 16
  Mid-diastolic murmur, (284)
  Pulmonary artery systolic murmur, 256, 266, **300**
  Tricuspid third sound, (313)
  Tricuspid systolic murmur, lessened, 261, (284), 303
    „ „ „ no change, (301)
Presystolic murmur:
  A spurious, due, in a rapid beat, to the approximation of a mid-diastolic aortic murmur to the following first sound, 394
Pulmonary artery systolic murmur, 254: *see* Summary, p. 551
Pulmonary artery: its altered relationships in upward dilatation of the right ventricle, 251
Pulmonary orifice: its dilatation a cause of the absence of the pulmonary systolic murmur in great upward dilatation of the right ventricle, 271, (294), &c., 556

Pulsation:
  In upward dilatation of the right ventricle its special characters, 17
  Diagnostic value of pulsation in the outer part of the third left interspace, 19
  Diagnostic value of pulsation at sternal end of fourth and fifth left interspaces, 29, 40
  Pulsation in the jugular bulb: its importance, 133
  „ „ neck veins: diagnostic significance of it, 137
  To the right of the sternum, usually due to the right ventricle not to the auricle, 41
Pulse, radial: characteristic of extreme heart weakness, 59
  Collapsing in fatty right ventricle with strong left, (155), 159
  May be an uncertain guide in adult and later life, 35, 143
Pulse, venous: felt in jugular bulb behind clavicle, 133

RABAGLIATE, DR., on muscular pains and tenderness, 478
Reduplication of the heart sounds, *see* Sounds of the Heart
  „ spurious, of the first sound may be due in a rapid heart to approximation of a third sound to the succeeding first sound, *vide* p. 394
  „ spurious, of second sound at the apex, *see* Third Sound
Referred pain in dilatation of the deep thigh-veins, 462
Relative percussion dullness, *see* Percussion, 21
  „ overstrain, as distinct from 'absolute,' 310, 343
Reservoirs:
  Auriculo-venous, a compensation reservoir for the right ventricle, 2, 584
  None for the left ventricle, 2
  Elastic one to lessen the strain of the ventricular contraction, constituted by first part of the aorta and of the pulmonary artery, 74
Reverberation as a method of diagnosis where the stomach is concerned, 164, 480
Reynaud's disease, symptoms of, due to colon dyspepsia, (128)
Rheumatism: its influence on cardiac distensibility, 11, 343
Right border of the cardiac dullness: its position in anæmic heart failure, 239
  Statistics, 354
RIGHT VENTRICLE (*see also* Ventricle)
  Dilatation of, *see* Essay VII, p. 329; and Summary, pp. 546 to 561
  Adolescent or upward type of dilatation, 9
    Its pathological aspects, 233, 247, 546
    Its clinical aspects, 12, 547
    Its various types, 289, 548
    The usual one, 289; mainly upwards, 290
    Mainly involving the conus arteriosus, 292, (293), (294)
    Mainly outwards to the left, 298
  Adult type of dilatation, 31, 557

Right ventricular overaction: its value as a measure of left ventricular failure, 47
  Its diagnostic value in later life, 86
  Its absence in severe pneumonia showing dangerous myocardial weakness, (98), (105)
  Mid-diastolic murmur, 282
  Third sound, 282
'Rigidity' of the heart wall in later life, definition of the term, **76**
  Difficulties in diagnosis due to it, 34, 79
  Mechanical elastic recoil due to it, 53, 80
  Its results in moderately strong hearts, **86**
  „ „ in very weak hearts, **91**
  Its clinical estimation, 147
  A case of weak heart showing its absence, (65), (157)
Rigidity of the first part of the aorta, diagnostic difficulties caused by it, 74, 109

Rolliston, Dr. on negative intraventricular pressure, 412
Rotation, after-sensation following it, is abnormal in vertigo of non-cardiac origin, 121
  Of the heart when displaced, 379

Sansom, Dr., on the third sound of the heart, 407, 427
Segmental pain in dilated deep thigh-veins, 462
Semicircular canals, hyperæsthesia of, a useful sign in the differential diagnosis of faintness and vertigo, 121, (123)
Small heart, a sign of myocardial weakness in distensible hearts, 6, **54**
Specific gravity of the blood: its minimal level determinable, 180
Sphygmomanometer: dyspnœa on its application, a sign of compensatory emptiness of the veins, 140
Sphygmo-oscillometer: its value in determining the meaning of high blood pressures, 144
Starvation causing compensatory diminution of the blood volume, 169
Sternum, the percussion of, in relation to the cardiac dullness, 25

Sounds of the Heart:
    Three sounds may mark the commencement of the three phases of the cardiac cycle, 399, 428
  Left ventricular first sound:
    Its accentuation a useful guide to the increased ventricular activity due to raised blood pressure, &c.
    An absence of accentuation in spite of increased arterial resistance is a sign of myocardial weakness, 146
    Its accentuation a compensatory phenomenon in mitral stenosis, 390, 405, **418**
    Its feebleness a compensatory phenomenon in aortic regurgitation, 409
      ,, ,, a sign of myocardial weakness, 96
    If short and sharp a sign of heart weakness, 96
  Right ventricular first sound:
    Its accentuation a useful guide to the amount of increased work done by the ventricle, 42
      In failing compensation in valvular disease, 420
      In embarrassment of the left ventricle in overstrain, &c., 47
      In anæmia, 244
    Absence of accentuation when the pulmonary circulation is embarrassed, a sign of myocardial weakness, 42
      Normal first sound in severe pneumonia a sign of heart weakness, (98), (105)
  Reduplication of the first sound:
    Spurious reduplication can result when in a rapid heart, a third sound is approximated to the following first sound, as with mid-diastolic murmur, 394,
  Second sound:
    Aortic accentuated in mitral stenosis owing to increased ventricular aspiration, 418, 423
    Pulmonary second sound, **550**
      Tables showing its loudness in valvular disease, &c., 421, 423, 424, &c.
      Its measured loudness in valvular disease when compensation is good and when imperfect, 423, 424
      Its apparent loudness in compensated mitral stenosis not a true accentuation, 392
      Accentuation in failing compensation, 422, 423
        ,, ,, anæmic heart failure, 415, 421
    Reduplication of the second sound, **550**
      Pulmonary half precedes the aortic in anæmic heart failure and in left ventricular overstrain, &c., 244, (297)
      Diagnostic importance of aortic half preceding the pulmonary half in right ventricular failure secondary to high arterial resistance

SOUNDS OF THE HEART (*continued*):
  THIRD SOUND OF THE HEART, 282, 340, **395**, 407, **427**
    Occurs at the commencement of the third or relaxation phase of the heart, one-tenth of a second after the second sound, 399, 428
    Due to the movement of the suddenly relaxed segments of an auriculo-ventricular valve if at the moment of relaxation there be any blood flowing through the valve orifice, 396, 431
    In the normal heart the flow has ceased prior to relaxation and normally no third sound occurs, 396, 430
    A third sound gives evidence of prolongation of the flow from the auricle into the ventricle: *vide* Mid-diastolic Murmur, 396, 430
      In mitral stenosis, 431
      When left auricle more dilated than left ventricle, 431
      In slight degrees of left ventricular failure, 431
        ,, ,, right ,, ,, in anæmia, 282, 396, (441)
        ,, ,, ,, ,, ,, in overstrain, (313)
      Cases showing character, &c., of third sound, (283), (284), &c.
    Possibly sometimes due to a preliminary closing of the auriculo-ventricular valve at the end of the expansion phase, 396
    Effect of change of posture on it, (284), (313)

Suction recoil wave in the cardiogram, 407, (428)
Suction by the ventricles (*see* Aspiration), 52, 79, &c.
'Superficial' percussion dullness, *see* Absolute Dullness, 21
Superior vena cava: its anatomical relationship to the first part of the aorta the cause of the ventricular wave in the venous pulse, 512
  ,, ,, ,, its dilatation the cause of the superior vena cava murmur, 44, 218, 561
Supra-clavicular fossæ, their fullness or hollowness, of diagnostic value in heart failure, 94, 134, 139
Syncope, pallor in, 450

TACHYCARDIA as a cardiac habit, its possibility, 325
Thigh veins, dilatation of, a cause of nervous symptoms, 459
Third sound of the heart: *vide* Sounds of the Heart, 427
  ,, ,, ,, its presence making three diastolic sounds audible, (284), (286), &c.
  ,, ,, ,, its presence a means of localising the interventricular septum, 284
Thoracic viscera diminish in bulk when volume of blood in circulation is compensatorily diminished, 163, 178
Tonic muscular contraction of:
  The right auricle, 500, 507
    In relation to tricuspid regurgitation, 38, 503, 509
  The great veins, 503, 507
  The right ventricle during its relaxation phase, 505
Trachea: its lateral displacement in collapse of one lung, owing to a displacement of the mediastinum, 380, fig. 140
Tracings from the veins of the neck, 135, 489
Transverse contraction of muscular fibres, 52, 384, 400
Tricuspid regurgitation, phenomena of, 37, 217, 239, 280, 338: *vide* Summary, p. 559

VALVULAR disease:
  Heart failure in, 562
  Early diagnosis of muscular failure in, 62, 110
  Estimate of rigidity of the heart wall in, 150
  Compensation in, 412

Valves of veins of neck, their physiological functions, 587
  Murmur due to their incompetence in tricuspid regurgitation, 219, (291)
Valves of the heart,
    Theory of compensation in disease of the mitral valve, 412
    „ „ „ „ aortic valves, 347
Vasomotor angina, (127), (130)
Vasomotor disturbance of reflex origin in anæmia, 292
„ „ a cause of intermittent claudication, 472, (475), (476)
Veins of thigh, severe discomfort due to pressure of dilated deep veins on the nerve trunks, 459
Veins of the neck:
  False pulsation in, 15, 39
  True pulsation, 16, 39
  Their emptiness in cardiac embarrassment a sign of myocardial weakness, 61, (106), 134, 138
  Diagnostic points connected with them given in tabular form, 137
  A visible diastolic wave in them is a sign of rigidity of the right ventricle, 398
  Importance of noting their condition in the heart failure of later life, 132
Vena azygos in relation to the auriculo-venous reservoir, 584, 588, 590, 592
Venous pulse in the jugular bulb, 133
  A true measure of the activity of the right ventricle, 134
Venous valves of the subclavian and jugular veins,
  Their importance as a part of the cardiac mechanism, 587
  Their incompetence a cause of pulsation in the neck and sometimes of a murmur, 39, 135, 219, (291)
Ventricles of the heart:
  Right more easily studied than the left, 2
  Apportionment of strain between the right and left ventricles, 2, 308, 545
    „ „ in adolescence, 5, 11
    „ „ in adult and later life, 31
  Diagnostic importance of slight degrees of overaction of the right as evidence of the early failure of the left, 46
  Dilatation of, *see* Dilatation
  Theory of ventricular dilatation, 9, 31
  Tonic activity of the right ventricle during its relaxation phase, 510
Ventricular expansion, *see* Expansion
  Evidence of the force developed by ventricular expansion, 436
  Compensatory dilatation of the left ventricle by its own aspiratory force 436
  Left ventricle sensitive to mechanical stimulation by over-distension, 497
Vertigo and faintness of labyrinthine origin simulating heart failure, 121 (123)
Vertigo of non-cardiac origin associated with hyperæsthesia of the semicircular canals, (123)

ZINC and starch dusting powder a means of writing upon the skin with one of the softer metals, 198

PRINTED BY
SPOTTISWOODE AND CO. LTD., COLCHESTER
LONDON AND ETON

COUNTWAY LIBRARY
HC 1H1I N

15.E.205.
The early diagnosis of heart fa1915
Countway Library BEP5151
3 2044 045 733 169